

COLUMBIA LIBRARIES OFF-SITE

HEALTH SCIENCES STANDARD



HX00044288

RJ460 F'77

Columbia University
in the City of New York

School of Dental and Oral Surgery



Reference Library



Digitized by the Internet Archive
in 2010 with funding from
Open Knowledge Commons

<http://www.archive.org/details/diseasesofmouthi00forc>

THE

DISEASES OF THE MOUTH

IN CHILDREN

(NON-SURGICAL).

BY

F. FORCHHEIMER, M.D.,

PROFESSOR OF PHYSIOLOGY AND CLINICAL DISEASES OF CHILDREN, MEDICAL COLLEGE OF OHIO;
MEMBER OF ASSOCIATION OF AMERICAN PHYSICIANS AND AMERICAN
PEDIATRIC SOCIETY, ETC.

PHILADELPHIA:
J. B. LIPPINCOTT COMPANY.
1892.

Dent
27-24054

Copyright, 1891, by J. B. LIPPINCOTT COMPANY.

RJ460
F17

PREFACE.

THE contents of this little book were first published in the form of a series of articles in the *Archives of Pediatrics*. Since then much has been done on the subject, and in most of the articles it has been found necessary to make additions and revisions.

The principal object of the work has been to bring together the facts in connection with diseases of the mouth in children, which has never been done before in the English language. For years the author has been preparing himself for this work by collecting clinical material, and his work will not have been in vain if he succeeds in helping to clear up the confusion that exists in English pediatric literature.

The importance of the subject dealt with will be recognized on all hands ; but the fact that the American Pediatric Association appointed a committee to make suggestions for an acceptable and universal nomenclature of the diseases of the mouth only emphasizes this statement. One of the principal motives for the republication of the articles was to give the medical student a systematic course which would give to him a working basis for his usefulness as a practitioner.

F. FORCHHEIMER, M.D.

CINCINNATI, October 7, 1891.

CONTENTS.

Introduction, 9-18. History, 9. Histology and Physiology, 10-12.
General Etiology, 12-13. Examination of the Mouth in Children,
13. General Treatment, 14-16. Classification, 17-18.

CHAPTER I.

Stomatitis Catarrhalis, 19-32. Etiology, 19-22. Pathological Anatomy
and Symptomatology, 22-28. Prognosis, 28-29. Treatment, 29-32.

CHAPTER II.

Stomatitis Aphthosa, 33-43. Historical Development of the Subject,
33-34. Definition, 34-36. Etiology, 36-39. Symptomatology, 39-
41. Prognosis, 41-42. Treatment, 42-43. Bednar's Aphthæ, 43-45.

CHAPTER III.

Stomatitis Mycosa, 46-67. History, General, 46-47; of the Fungus, 47-
53. Etiology and Pathology, 53-57. Pathological Anatomy, 58-59.
Symptomatology, 59-64. Prognosis, 64. Treatment, 65-67.

CHAPTER IV.

Stomatitis Ulcerosa, 68-86. History, 68-69. Etiology, 69-76. Patho-
logical Anatomy, 76-77. Symptomatology, 77-83. Prognosis, 83.
Treatment, 83-86.

CHAPTER V.

Stomatitis Gangrenosa, 87-98. History, 87. Frequency of the Disease,
87. Etiology, 88-91. Pathological Anatomy, 91. Symptomatology,
General, 91-92; Local, 92-95. Prognosis, 95. Treatment, Prophylac-
tic, 95; General, 96; Local, 96-98.

CHAPTER VI.

Stomatitis Crouposa—Stomatitis Diphtheritica, 99-104. Stomatitis
Orouposa, 99-100. Stomatitis Diphtheritica, 100. Symptomatology,
102-103. Prognosis, 103. Treatment, 104.

CHAPTER VII.

Stomatitis Syphilitica, 105-122. Syphilitic Manifestations in the Mouth; Fissures, 106-107; Papules, Plaques, 107; upon the Tongue, 107-109; Geographic Tongue, its Non-Syphilitic Nature, 109-114. Syphilitic Teeth, 114-117. Hutchinson's Teeth, 117-118. Fournier, 118. Treatment, 120-122. Stomatitis Leptostricia, 122-123.

CHAPTER VIII.

Dentition, 124-167. The Older Writers, 126-129. The Authors of the Nineteenth Century, 129-130. The Development of Teeth, 130-132. The Time and Order of the Appearance of Teeth, 133-136. Retardation of Teething, 136-137. Premature Teeth, 138-141. Late Teeth, 142-144. Permanent Teeth, 144. Symptomatology of Dentitio Difficilis, Local Symptoms, 144-147; General Symptoms, 147-154. Treatment, 154. Gum Lancing, 156-167. Conclusions, 167.

CHAPTER IX.

The Tongue and the Mouth in Disease of Remote Parts, 168-178. Changes in Shape and Size of the Tongue, 170. Changes in Color, 170-172. Coating of the Tongue, 172-175. Ulcers, 175. The Tongue and Mouth in the Acute Exanthemata, 176-178; in Disease of the Nervous System, 178.

CHAPTER X.

Parotitis, 179-180.

CHAPTER XI.

Epidemic Parotitis, 180-189. Etiology, 180-181. Symptomatology and Pathological Anatomy, 182-189. Complications and Treatment, 190-191.

DISEASES OF THE MOUTH

(NON-SURGICAL).

IT is difficult to find a subject in diseases of children upon which so much confusion exists as upon the above. There are many reasons for this. The historical development of the subject has been slow; indeed, very little clearing has been done up to within the last fifteen or twenty years; then, a term first used by Hippocrates (*aphthæ*) has caused considerable confusion. Finally, these forms of disease have suffered, in common with all diseases of children, on account of inattention, consequently lack of observation, or observation made in the direction of some preconceived view or theory only. Hippocrates and Galen and their followers first used the term *aphthæ*, and, by degrees, every trouble that took place in the mouth was called *aphthous*. The distinctions and subdivisions made were in some instances simply ludicrous. It was not until the time of Bretonneau that diphtheritic sore mouth was separated from *aphthæ*, and some authors drew their lines of subdivision so finely that they could make a great number of varieties of *aphthæ* (16 Sagar). The result of this was that everything was so completely confused that one writer failed to understand the other. Even if our present textbooks are carefully examined into, notably those on practice, it will be seen how lamentably weak, in nearly all instances, the chapter or chapters on the diseases of the mouth are, the omissions not to be taken into consideration at all. A great

deal of this must be due to the fact that misunderstandings are caused by the very inaccurate and confusing nomenclature still in use. In considering the non-surgical diseases of the mouth we can divide up the subject into two broad subdivisions to begin with: first, the diseases affecting those parts within the mouth; secondly, those diseases affecting the organs outside the mouth whose physiological functions are carried on in the mouth. Under the first heading come all the affections of the mucous membrane, its various layers and its glands, the teeth and the tongue; under the second, those of the salivary glands and their ducts. Before going on to the separate subdivisions and their discussion, it is necessary to take a glance at the physiological processes going on within the mouth of a child. From a physiological stand-point, the mouth of a newly-born child must be looked upon as a passage-way for food endowed with organs of suction. Although ptyalin has been found in infusions from the salivary glands of the newly born (Zweifel, Korownin), the fact remains that, for digestive purposes, the mouth can be practically excluded. The reason is to be found in the often-repeated observation that before dentition the mouth of the child contains very little saliva. If we inspect the mouth of a very young child—up to three or four months, seldom later—we will find the mucous membrane comparatively dry, the tongue always more or less coated and dry, and of a peculiar color and reaction to reflected light. The coating is sometimes found evenly distributed over the surface, but more commonly it is especially developed where muscular activity would have the least effect upon disturbing the epithelial layer; the edges, tip, and centre would therefore have a smaller deposit than the other parts of the tongue. If the scrapings of such a tongue be examined under the microscope, it will be seen to be made up principally of food-remnants and epithelial cells, the former predominating. In the healthy adult the saliva is poured

out upon the least excitation, central or reflex, therefore the tongue is usually clean, as the mouth is constantly washed by fluid; but in infants this rarely takes place, even after the administration of sialagogues (*jaborandi* perhaps excepted), therefore the tongue is coated and the mouth dry. When we take into consideration how much value is still attached to the appearance of the tongue in disease, this fact is worthy of consideration. Usually the salivary glands begin to be called into activity before the teeth make their appearance,—the time varies very much, sometimes as much as three or four months elapsing. It is impossible to say whether this is due simply to reflex activity or progress in development of the glands. It is certain that if the mouth of a newly-born infant is irritated mechanically, very little if any saliva will flow. Again, if we irritate the mouth of an infant beginning to produce saliva, the increase will be hardly appreciable. If there is, then, a reflex mechanism at work in these instances, it must be an incomplete one, either as to the sensitive nerve, the centre, or the secretory nerves. The secretory nerves do not seem at fault, for, first, there is a quantity of saliva secreted, and, secondly, if the diastatic power of this saliva be tested in the usual manner, it will be found to be good; perhaps not always as rapid as with adult saliva, but sufficiently so to show that ptyalin is present in adequate quantity. As far as concerns the action of the salivary centre, at the origin of the seventh and ninth cranial nerves in the medulla, we do not possess any facts which could lead us to suppose that it acts differently in infants than in the adult; yet this might be possible, as the brain of an infant does perform different and incomplete functions from that of an adult. The experience of Mischterlich—no irritation, no saliva—does not help us out, for, if any irritant be put into the mouth of an infant, reaction in the form of motion will take place and yet no saliva may follow. Experiments and observations in this direction would

be very desirable, not only on account of clearing up those processes, but also because of the important rôle the nerve-mechanism of the mouth has always played in infant etiology. When the flow of saliva has been started, the mouth of the infant does not change its character to an adult mouth immediately, for the simple reason that most of the saliva flows from the mouth, and not through it. The tongue, especially, remains as it was, as the saliva from the parotids flows along the cheeks, between them and the partly-opened mouth, and that from the submaxillary and sublingual glands over the lips. Very little saliva is swallowed, so that its digestive activity, and with it that of the pancreas, must still be very limited,—a fact of great importance in dietetics. The nature of the food—coagulability, adhesibility, fluidity—must also be taken into consideration in estimating the appearance of a child's mouth, so that peculiarities are common long after the salivary function has been thoroughly established. The appearance of the teeth marks an epoch in the development of the child, and, as has been the case with all physiological processes, most, if not all, of the ailments of childhood have been ascribed to it. The question of teeth and teething is of such value to us as physicians that it will be discussed separately.

On the subject of etiology our lack of knowledge is still great, although much has been done within late years. Nowhere in the human organism do we find so admirable a field for the development of lower forms of life as in the mouth of an infant. The great number of forms present (Miller describes twenty-five varieties) has undoubtedly made the work of bacteriologists doubly hard, and in some instances must have made it futile; but, with advanced methods and repeated, patient efforts, very much more will be accomplished. As far as general symptomatology is concerned there is but one symptom that need be specially dwelt upon, and that is pain. This is present in nearly every form of sore mouth, and in some it

is the prominent symptom. It is a good rule to follow, that when an infant is suffering with pain which cannot be localized, to examine its clothing, its mouth and throat, and its ears. Those possessing clinical experience have seen children who have been crying for days, who have perhaps been treated by physicians, taken opiates or chloral, in whom an examination reveals stomatitis ulcerosa. The diagnosis made, the whole picture will clear up in twenty-four hours upon proper treatment. A case of this description is the more remarkable because the mouth of a child can be so readily examined. It is not necessary to carry a set of instruments for the purpose; all that is required is good light and, if necessary, a separation of the lips or the holding down of the tongue with a spoon. A tongue-depressor ought not to be used, for several reasons: a little child will always be more frightened at an instrument with which it is not familiar than at a spoon, and, secondly, a tongue-depressor may be the means of carrying infection if not kept aseptic. The latter, although theoretically the case, in busy practice is apt to be neglected, and damage is very easily done to an already inflamed mouth. On account of the facility with which the mouth is examined, the purely clinical aspect of our subject is perhaps best understood, but for the same reason most often neglected, as we are apt to overlook those things that are nearest. It is necessary to call attention to the fact that in the treatment of all diseases of the mouth cleanliness is of the highest importance, although the experiments of Fischer seem to show that even this may do harm. It is astonishing to see how the idea of cleanliness varies, both with the laity and physicians. With the latter it will only be a matter of time until perfect cleanliness is thoroughly understood and appreciated. It is not beneath the dignity of a physician to teach his patients how to cleanse. Roughness, too, should be avoided in treating sore mouths; not to mention the pain that is given, we do absolute harm by using mechani-

cal violence. In but one form of stomatitis is it necessary to remove anything; in all the rest, applications made by the gentlest means will give the best results. As was first shown by Rajewsky in diphtheria, all inflamed mucous membranes are more susceptible to infection than healthy ones. If we are dealing with infectious processes, and this is the case with many of the affections of the mouth, it will be seen how injury to the mucous membrane can only lead to an extension of the process. Hunt and West were the first to use chlorate of potassium in the treatment of diseases of the mouth, and since that time the remedy has been used by the profession, and in some cases has been regarded as a specific. This remedy has deservedly retained its place upon our list of drugs; and it acts equally well if applied locally or by the stomach,—a fact of great importance, especially in the treatment of younger children. It appears in the saliva, when taken internally, after a very short time (from five to ten minutes), and its secretion continues for some time, so that it is best given in small doses at short intervals. Its use, however, is not unattended by danger,—a fact which Jacobi was the first to call attention to,—as it affects the secretion of urine, and may even produce anatomical lesions in the kidneys. Jaederholm (1876) was the first to call attention to the fact that methæmoglobin was produced by the action of potassium chlorate upon blood, and Marchand (1879) followed by showing the connection between this fact and the symptoms observed in cases of poisoning with this drug. He, as well as Jacobi, describes the lesions in the kidneys, but he lays most stress upon the change in the blood, and Mering, in a very important memoir ("Das Chlor-saure Kali," Berlin, 1885), contributes further knowledge in that he explains some of the concomitants necessary to make a comparatively small dose lethal. He claims that a small quantity of potassium chlorate is decomposed in the blood; under all circumstances, by far the greatest quantity leaving the sys-

tem unchanged by the kidneys and the salivary glands. The result of this decomposition is methæmoglobin, a very small quantity of which does no harm in the circulation. This methæmoglobin leaves the circulation by being excreted in the urine. Poisoning by potassium chlorate may act in two ways; first, by destroying the oxygen-carrying function of the blood by a wholesale conversion of oxyhæmoglobin to methæmoglobin; secondly, by causing a choking up of the kidneys with methæmoglobin and detritus from the destroyed red corpuscles. In the first instance a large dose has been taken, and death follows in a comparatively short time; here the prominent post-mortem evidence will be the change in the physical appearance of the blood. In the second instance large or comparatively large doses have been taken for some time, and death follows in from two to fourteen days; in these cases both the blood-changes and the changes in the viscera, kidneys, and spleen will be observed, and Mering calls attention to the fact that in all those conditions in which there exists in the blood an increase of the acid phosphates or carbonic dioxide, or in which the alkalinity of the blood is only slightly diminished, the toxic effects of potassium chlorate are enormously increased. Marchand was the first to insist that use of potassium chlorate ought to be abandoned in children, and we see this statement repeated by Landerer, who recommends that its internal administration be given up entirely, especially in children (*Deutsches Archiv für Klinische Medicin*, xlvi., 1890, p. 125). The answer may be given that, as soon as a substitute is found for the potassium chlorate, this will certainly occur, especially in very young children. At the present, however, it seems to the author that more harm would be done by not prescribing this drug than by administering it judiciously. In doses to be recommended, and with the precautions mentioned before, its use is as safe as that of any of the drugs possessing toxic effects. The fact must not be lost sight of,

however, that potassium chlorate may act as a violent poison. In order to prevent this, two things are necessary: not to give too large doses of the drug, and, secondly, to impress upon the attendants the necessity of watching the child in certain directions. It has been the result of the author's observation that the symptoms of chlorate of potassium poisoning do not develop suddenly, but are usually preceded by symptoms which will give plenty of time to prevent dangerous consequences if the remedy be stopped. These symptoms are diminution or cessation of formation of urine, and great drowsiness,—they usually go together, and are to be looked upon as a warning. In some children, especially infants, chlorate of potassium will act like a large dose of opium.

It is proper, in this connection, to call attention to the fact that diseases of the mouth, especially in infants, and more so in some forms than in others, are not to be treated as purely local affections. If we abstract entirely from the recognized fact that some of these diseases have a constitutional origin, and that many more are provoked by general predisposing causes, there is still left a factor which must make us very cautious in observing the general effects of these maladies. Especial reference is made to the connection that exists between the mouth and the rest of the alimentary canal. It seems that under normal conditions the gastric juice is in a condition to destroy most of the lower pathogenic forms of life, but if we consider how finely balanced is the digestive process in children, and how little it takes to convert eupepsia into dyspepsia, it will be seen how disastrous may become the swallowing of large quantities of saliva and mucus, if by their chemical interference only. Now, add to this lower forms of life in the saliva, which, under the changed conditions in the stomach, cannot be destroyed, the effects will be still more marked. It is not surprising, therefore, that a great many authors have sought a causal connection between diseases of the

stomach and diseases of the mouth,—as disturbances of digestion are so common with disturbances in the mouth,—but that the primary cause was referred to the stomach was rather remarkable. It is not an infrequent experience to see a case of stomatitis ulcerosa treated with strict diet, which cannot do harm until pushed to extremes, because “the sore mouth comes from the stomach.” The author has seen children, with conditions of the mouth probably congenital and possibly lasting during the lifetime of the patient, put upon rigid diet, made to take pepsin, arsenic, or what not by most reputable practitioners, only because all diseases of the mouth have their origin in the stomach. It is unnecessary to add that the results of such treatment were *nil*, and a few local applications were sufficient to alleviate those symptoms which brought the patient to the physician. As will be seen, the term stomatitis has been retained to mean sore mouth. Strictly speaking, this is incorrect, as stomatitis means an inflammation of the mouth; but classification becomes very much easier by retaining this term, which is used by nearly all nations, and therefore it facilitates memory by bringing all forms together under one heading. If the mind can group things together in this way, differential diagnosis also becomes easier, and the attention will always be called to the whole group, from which the individual can be more readily selected. The great objection to this classification is that in order to make it complete so many species must be made that it becomes bulky and we counteract the benefits before mentioned. This can be prevented, however, by rejecting special names for those forms that are symptoms of general diseases, and which belong to the latter, as stomatitis scarlatinosa, stomatitis erysipelatosa, etc. The use of a Latin instead of an English term is certainly advisable, as it gives to all physicians of the world a common language, which, in these days of rapid interchange of thought, is highly important and time-saving.

In the classification which we will follow we make the following subdivisions :

- I. Stomatitis catarrhalis.
- II. Stomatitis aphthosa.
- III. Stomatitis ulcerosa.
- IV. Stomatitis mycosa.
- V. Stomatitis gangrenosa.
- VI. Stomatitis crouposa.
Stomatitis diphtheritica.
- VII. Stomatitis syphilitica.

I.

STOMATITIS CATARRHALIS.

THIS form of trouble has also been called simple stomatitis. By some of the English authors it has been described under a common heading with follicular and aphthous sore mouth, from which it can be, however, most readily distinguished. Two subdivisions can be made: first, a local; secondly, a general catarrhal stomatitis.

Etiology.—Catarrhal stomatitis may be produced in various ways. For its production it is necessary to consider two things: first, an irritant; secondly, the mucous membrane. The irritation may consist of very many agents; it may be mechanical, thermal, chemical, or some lower form of life which acts either mechanically or chemically. The teeth have been looked upon as the most common mechanical agent in producing stomatitis. While there can be no doubt about the fact that when a tooth is about to appear there is more or less injection and swelling of the gums, yet in a healthy child this alone would never be sufficient to produce a general stomatitis. There are various well-marked lesions which are produced by teething, and which will be considered at some future time, but for the production of a general catarrhal stomatitis the second etiological factor mentioned above must be present. Lack of cleanliness in the mouth is a well-recognized cause for the trouble under consideration. This may be causative in various directions, quality and quantity of food being the most important, and their action being chemical and mechanical. A child fed upon food which is in fermentation, which has a very acid reaction, or particles of which are apt to remain in the mouth, will suffer more or less, depending upon the intensity of the irritation. The same can be said for food introduced at too high a temperature. Many mothers are in

the habit of feeding their children with milk which is too warm, and in tea-drinking countries, like England, it is not uncommon to find even more serious affections follow the introduction of this beverage when too hot (retro-pharyngeal abscess, Bokai). An increase in quantity of food will produce stomatitis in an indirect way by causing trouble with the whole alimentary tract. Any weak chemical irritant acting for a long time, or a comparatively strong one when swallowed rapidly and mixed with much saliva, is apt to produce this affection. It is difficult to conceive of eructations of sour-stomach contents, when not habitual, causing a sore mouth, yet vomiting, when extending over a long time, is apt to be followed by it; although here, again, it is difficult to say whether the cause which has produced the dyspepsia has not also produced the sore mouth. As far as lower forms of life are concerned, it is impossible at the present time to say positively that any of them can be looked upon as causative. To my knowledge, no experiments have as yet been made in this connection. There is one fact, however, known long before the days of the culture-tube, and which points in this direction. It has been thoroughly understood for the last ten years, at least, that nearly all forms of sore mouth are preceded by a stomatitis catarrhalis. For this an explanation can be found in two ways only: either the same cause is at work for stomatitis catarrhalis and the other forms, or it is necessary that the mucous membrane be in a proper condition to be affected by the poisons of the other forms only after it has been first made catarrhal. The latter is the view held by very many authorities, it being most commonly expressed by the statement that the child is suffering from malnutrition, a dyscrasia, scrofula, or what not. One quotation will suffice: "Follicular (or simple) stomatitis is not a serious complaint, though it indicates a weak state of health and a faulty nutrition" (W. Fairlie Clarke in Quain's "Dictionary"). While it is difficult to disprove such a statement

as the latter, it will be seen, upon closer investigation, that the stomatitis and the intestinal catarrh or dyspepsia accompanying it is the cause of the "weak state of health," and not the result. However, that "a faulty nutrition," or whatever it may be called, has a decided effect upon producing stomatitis will be pointed out later. Whether or no *oedium albicans*, the various pathogenic schizomycetes, also produce the combination of symptoms which we call stomatitis, as well as dyspepsia, is a thing to be decided by direct experimentation, although there are a great many facts in bacteriology which would lead us to suppose that this might be possible. If we accept one view or the other, the subject of catarrhal stomatitis assumes great importance when we recognize that this disease may lead to others of a much more serious nature, which may be prevented by proper treatment. As far as the second etiological factor, the mucous membrane itself, is concerned, it must be remembered that it is in direct continuous connection with the mucous membrane of the nose and the pharynx. A catarrh of any one of these membranes may extend itself to the mouth; this does occur, but not frequently. Finally, a non-healthy mucous membrane, whose nutrition is impaired by disturbances in circulation either in the blood- or lymph-vessels, or whose blood-supply is otherwise not good, would naturally form a better soil for the implantation of pathogenic causes than a healthy membrane. From such a mucous membrane it would also be more difficult to remove such causative agents, as it would require less time for them to gain a foothold. A so-called scrofulous child would be more apt to suffer with sore mouth than a healthy one (for mechanism see Buck's "Hand-Book," article "Tuberculosis of Glands"). The same would be true for a child whose resistance-power is reduced by any of the febrile diseases which last for a considerable length of time—typhoid fever, malaria, the acute exanthemata—or by chronic intestinal diseases. It will be seen, then, that the con-

dition of the mucous membrane is of the highest importance in the prevention of stomatitis; and, undoubtedly, with a little care a great many diseases—diphtheria, for instance—could be prevented if apparently trifling abnormalities would be looked after more closely. Bad cases of harelip or cleft-palate will cause more or less chronic stomatitis, as the air is constantly brought into contact with the mucous membrane, which ought otherwise to be closed off, at least temporarily. In the localized form the causes are, in general, the same as those for the other form, only not so extensive in their action. Most commonly we will find some quite localized irritation,—a sharp tooth or pus flowing from a chronic perialveolar abscess,—which keeps up a condition of inflammation. Again, it may be some article of food or a method of feeding. In some parts of Germany that ingenious device called a Lutsch-beutel always succeeds in getting up a stomatitis, either local or general. With us some of the beautiful apparatuses invented to facilitate teething, especially when they are rolled about the floor or contain materials which can be fermented or are otherwise unclean, succeed admirably in accomplishing our end. At all events, the cause can be readily removed in most instances, and, unless harm has been done in other directions, the patient rapidly recovers.

Pathological anatomy and symptomatology will be considered together, as all observations can be made during life. Catarrhal stomatitis has no favorite starting-place, nor can we say that it limits itself to any especial locality in the mouth, except in the rarer localized form, in which the appearances of the mouth do not differ from those of the generalized form, if we take extensy of affection into consideration. The tongue, the soft and hard palate, the cheeks, the buccal surface of the lips, and the mucous membrane covering the jaws are all affected. If we look into the mouth of a child with simple stomatitis we see various grades of change: first, the erythe-

matous ; second, the true catarrhal. Further subdivision is unnecessary, as in all other forms, especially in the one by many authors called follicular, all the changes are the logical outcome of an inflammatory process upon a peculiarly constructed mucous membrane ; just as acne vulgaris is found upon the skin. In the erythematous form the whole mucous membrane of the mouth takes upon itself a more or less deep red color. The process can be looked upon as the result of irritation which is not sufficiently intense to be followed by inflammatory reaction. There exists, then, hyperæmia only, which, as a rule, is of such a transitory nature that deeper changes do not follow. The hyperæmia may be so well marked—and this is often the case in newly-born infants—that rhesis occurs, followed by slight hemorrhages, or red corpuscles may be forced out into the lymphatic spaces of the subepithelial connective tissue, whose coloring-matter changed to hæmatoidin will give a distinct yellow tint to the mucous membrane. This is especially the case over the hard and soft palate, a process analogous to the one in the skin in icterus neonatorum. The condition of erythema of the mouth may be looked upon as normal in the newly born, and requires no attention, as it disappears after the first week of life, rarely lasting longer. This process being a very superficial one there are no changes in the glands, either of the mucous membrane or of the lymphatics. Therefore the functions of the mucous membrane are not interfered with. As a rule, there is no hypersecretion, but, on the contrary, there exists more or less dryness of the mouth, which must find its explanation in the fact that the temporary nutrition of the epithelial coating is interfered with. There is one form of erythema to which especial attention must be called. It is found in pertussis and, as far as the tongue is concerned, in measles.

The appearance of the mouth in the acute exanthemata will be discussed in another place, but the following description

also holds good for the tongue in measles. If we look at the mouth of a patient suffering with pertussis, perhaps the most striking thing to be observed is the blue color of the tongue and the rest of the oral cavity. The mechanism is simply the production of venous hyperæmia by the repeated attacks of coughing, which prevent the ready return of blood to the right side of the heart. In measles this bluish color is due partly to the cough, partly to the appearance of an eruption in the mouth, and in both conditions it sometimes helps in making a diagnosis. It is not, however, characteristic of either condition, as, like the ulcer of the frenulum linguæ of pertussis, it may exist with any cough that is persistent or comes in frequent attacks, like the cough of enlarged bronchial glands or of tracheitis; or it may exist in troubles of the respiratory or circulatory organs which prevent the blood from returning to the heart, or in which the blood is insufficiently aërated, as in catarrhal pneumonia, pleurisy with large effusion, insufficiency of the valves of the heart, etc. If we add that a slight rise of temperature in an infant will produce erythema of the mouth, and that sometimes lesions of the skin of an erythematous nature are accompanied by the same change in the mouth, we have said all that need be said of this trouble which may be of importance from a diagnostic stand-point, but hardly in any other direction. It is doubtful, indeed, whether a simple hyperæmia can be necessary to the development of any other disease of the mouth. The form described in connection with pertussis might be looked at in this direction, but in many cases the border-line of erythema and inflammation is overstepped, and then we are dealing with catarrhal stomatitis, which is of far greater importance in all directions. In individual cases it may become very difficult to say whether it is erythema or something more that we are dealing with, although, as a rule, the tissues which are involved will readily clear up the question.

In catarrhal stomatitis the lesions are so well marked that, with ordinary care in examination, it is difficult to overlook them. In this form of trouble we have, as a rule, all the symptoms of inflammation,—swelling, heat, pain,—which manifest themselves differently according to location. The whole lining of the mouth is red, there is hypersecretion after the process is well under way, and the temperature of the mouth is increased. If we examine carefully it will be seen that the mucous membrane lining the cheeks is puffy; if there are any teeth, it is marked by depressions where the swollen membrane presses upon them. The color of this part of the mouth, especially of the depressions, is paler than the rest of the mucous membrane, and it is not uncommon to find these little valleys surrounded by elevations whose contours are marked by dilated vessels. The slightest injury causes a rupture of these already weakened blood-vessels, so that slight hemorrhages or saliva mixed with blood are not infrequent. Over the hard palate the mucous membrane is not much swollen, for anatomical reasons; but the injection of the blood-vessels is well marked, sometimes general, at others more or less localized.

In older children the mucous membrane behind the upper incisor teeth is, as a rule, very puffy, although not very red, and very painful. In infants this part is also affected, but not to the same degree, yet not infrequently it takes upon itself a spongy appearance, although it does not appear faceted as in older children. The lips are swollen; if taken between the fingers they are more tense than normal, and their inner aspect is very much reddened. The surface of the mucous membrane is made uneven by small round prominences. These are the muciparous follicles whose ducts have become partially stopped up, or in which the secretion has accumulated so rapidly that the whole body of the gland is filled up. Sometimes there exists complete occlusion of the duct, then there follows an enormous dilatation of the gland, which manifests itself in the

production of a cyst. When this cyst is opened a small quantity of mucus is discharged, but the cyst is liable to refill, emptying itself by being broken from time to time, and always forming again unless active treatment is used. This is a comparatively rare complication, and, as a rule, the ordinary glandular involvement of simple stomatitis runs its course, even without the production of ulcerations.

On the other hand, slight epithelial abrasions over these swollen follicles or in other parts of the mucous membrane are by no means rare, even in infants, although they rarely lead to the involvement of the deeper layers. The tongue is at first covered with a dry whitish coating, quite uniform over the whole surface; as secretion increases this becomes more moist, and is washed off in places, usually about the edges. With this, the tongue—its upper surface at least—may be slightly swollen, and its color soon changes. The coating is no longer of a chalk-white, but grayish or even yellowish, and it may look as if the epithelial layer might be stripped off in a flake without detriment to the organ itself. This does not occur, however, as the process seems to affect the older cells only, rarely leaving the mucous membrane completely denuded, due undoubtedly to the fact that there is so much fluid present in the mouth. Through this coating the fungiform papillæ, very much swollen and injected, are visible. The tips of the filiform papillæ are involved in the general process going on in the epithelium, but their bases seem to remain intact even where the epithelium falls off, so that the tongue never has the appearance of a strawberry or the hilly, shaved syphilitic tongue. Where the epithelium is partly stripped off we have an intensely red color. The edges of the tongue are rounded off, and where there are teeth we find the same depressions noticed in the cheeks.

If with stomatitis catarrhalis there is associated a process accompanied by continuous fever (typhoid, remittens or the

exanthemata), we have all that has just been mentioned ; but after a few days the epithelium dries up and falls off, leaving a raw surface, sometimes fissured, the color of which varies greatly, principally on account of the quantity of blood present upon it. The whole mouth partakes to a greater or less extent in this change, producing the dry, cracked lips, the sordes upon the teeth, etc.

In all cases the lymphatic glands supplied by the mouth are more or less involved, and it is a safe rule to measure the grade of the stomatitis by the amount of enlargement there is in the lymphatics. There are mild forms of stomatitis catarrhalis that affect the patient very little ; sometimes, even, we are astonished to find a very extensive inflammation with very little general reaction on the part of the patient. As a rule, however, the patient complains of well-marked symptoms which alone will lead the initiated to localize the seat of trouble in the mouth. There is usually present more or less fever, rarely going very high, going down to normal in the morning and up to 101° - 102° F. (rectal) in the evening. In some children the temperature may go quite high (104° F.), and may require special attention.

The prominent symptoms of stomatitis are the manifestations of pain and the hypersecretion of saliva. The little patient, if an infant, goes at the breast with a good will, evidently hungry, takes one or two pulls, then suddenly lets go of the nipple and begins to cry. By means of a little coaxing the mother will be able to succeed in getting the little one to try again, but the same result follows, and, finally, the baby refuses absolutely to be put to the breast, preferring to remain hungry to suffering pain. In the intervals between feeding the child may be cross and fretful ; it may whine considerably, but does not cry out very much, as in some other forms of stomatitis. At the same time the child, if old enough, is drooling constantly, the saliva flows from its mouth freely, and

the mother is apt to be happy over the whole condition because she thinks her baby is teething. This increased flow of saliva produces irritation of the skin over the lower lip, the chin, sometimes the neck, and many an eczema is started up by stomatitis. Long after the irritant—the saliva—has been removed the eczema still remains, and may give rise to eczema in other parts of the body or universal eczema. Bohn states that the reaction of the saliva may be neutral, never alkaline; I have never been able to find any other reaction than an acid one. As has been pointed out in the introductory chapter, not every infant or child drools, so that this symptom is frequently absent.

The effect of a simple stomatitis upon the general condition of a child or infant may be *nil* or it may be of the severest nature, even costing the child its life. Just as a nasal catarrh may prove fatal, so a stomatitis may kill by preventing the child from taking its food,—*i.e.*, more or less directly. This manifestly is the rarer modus; but given a badly-nourished infant which becomes affected with a stomatitis, and two or three days of complete abstinence from food will be sufficient to reduce the vitality to such an extent that recovery is impossible. Or the stomatitis may produce dyspepsia, catarrhal conditions of the intestine, and death in this way. It is not uncommon to see dyspepsia set up as the result of bronchitis, a coryza, or a stomatitis due possibly to the swallowing of something coming from one of the affected mucous membranes. This “something” may be an increased amount of fluid or fluid containing an irritant; in either case followed by reaction, which causes dyspepsia. The fatal termination, again, is rare; but commonly do we find the child’s nutrition suffering, so that great care and attention are required to save the child’s life in attacks of other diseases.

It will be seen, therefore, that, *quoad vitam*, even this apparently trifling affection is of great importance. It may

be stated, furthermore, that, once a child has had general stomatitis catarrhalis, the least irritant will produce a partial or general return of the trouble, so that in badly-nourished marantic children the condition becomes chronic. In healthy children this is not the case, although in them a running down is apt to be followed by another attack, provided the external causes are present. Infants are more liable to this disease than older children, although in the latter it is by no means uncommon, being overlooked in them on account of absence of symptoms.

Treatment.—The importance of conscientious treatment is to be found in the fact that this disease may be the forerunner of other more serious troubles, as has already been pointed out. As a rule, a general catarrhal stomatitis runs a favorable course without any special treatment. Indeed, it must be taken for granted that this is the case in the great majority of instances, as the better class of children, only, are under such strict surveillance as to be placed under the care of a physician whenever a slight ailment exists. It is impossible to enter into such details as would cover the whole ground in each individual case; this is fortunately unnecessary, as after all the principles of treatment are the same whether applied to an inflammation of the mouth or any other part of the body.

Prophylaxis is highly important; with nurses we usually find the two extremes in the care of the mouth of infants. They are either oblivious of the necessity of looking after the mouth, or they treat it with such violence as to do more harm than good (see chapter on Bednar's aphthæ). The rough finger of the rougher nurse is used as the means for cleaning the mouth,—perhaps wrapped in a diaphonous handkerchief,—and this is pushed into the unoffending mouth, scraping away everything with which it comes into contact. Or the mouth is never examined at all, and, much to the surprise of every one, there develops suddenly a stomatitis of one kind or another.

Under normal circumstances, the mouth of every infant ought to be washed out several times daily with lukewarm water which has been previously boiled. A small wad of absorbent cotton, wrapped upon a smooth stick or wire, is as good a contrivance as any, as it insures cleanliness and, with reasonable care, is perfectly safe. For infants and restless children the cotton can be wrapped around the finger of the nurse. The rule must be laid down as absolute that the cotton must be replaced by a fresh piece every time the cleansing is done. A large camel's-hair brush is more convenient but not so safe, as far as being the possible carrier of infection. Mothers must be taught to regard cleanliness in the mouth as of the same importance as upon the external surface. For this reason they must be taught to keep their nipples in good condition, and if the child is brought up artificially, how to take care of all of the various articles necessary to artificial feeding. The quality and physical properties of the food, in the latter instance, must be especially dwelt upon, notably the temperature. In older children, the tooth-brush will frequently prevent attacks of localized stomatitis. All irritating substances which act as foreign bodies, such as sharp teeth, accumulations of so-called tartar, perialveolar abscesses, etc., must be treated and, if possible, removed. In the course of febrile affections much can be done to prevent affections of the mouth, which, unfortunately, are still too common. If the patient is old enough he can be taught to suck small pieces of linen which have been dipped into ice-water or in which small pieces of ice have been wrapped. The sordes, cracked tongue, etc., of continued fevers can be easily prevented by a moderate amount of care. If we are dealing with a young child or one delirious, frequent washing of the mouth with cold water will accomplish the end almost as readily. Either of these plans is both grateful and beneficial to the patient; grateful, especially, in that it relieves the thirst which is always present with high fever.

The treatment of the affection, once the cause is removed, is a very simple matter. All food must be given cold,—it causes less pain to the patient and reduces the swelling of the mucous membrane. If necessary the milk can be cooled by putting the vessel containing it into ice. This, however, is purely empirical, as some children with stomatitis catarrhalis bear their food better when it is quite warm. In children at the breast this falls away, of necessity, as it would be reprehensible to change the food on account of the benefit which might accrue by having its temperature reduced. The mouth must be gently washed, as often as possible. Cold water—ice-cold if necessary—which has first been boiled is, as a rule, sufficient. As lotions a great many substances have been used: boric acid (two- to three-per-cent. solution), sodium biborate (five to ten per cent.), zinc sulphate (one-half to one per cent.), salicylate of sodium, salol, etc. Most, if not all, are unnecessary except as to the probability of directions being followed more exactly when a mouth-wash is prescribed. The internal or external use of potassium chlorate is also unnecessary and, according to my experience, valueless in this form of trouble. Unless absolutely indicated, potassium chlorate ought not to be used, on account of the risks attending its administration; it does not seem necessary to take any risks in the treatment of a simple stomatitis. The most reliable of all medicaments is silver nitrate (one-half to one per cent.). If the stomatitis does not disappear in three or four days, the mouth ought to be pencilled with this weak solution of silver nitrate once a day. Before applying the solution the mouth must be carefully washed out with cold water. Whenever there is a loss of epithelium, or an ulcer, the mitigated stick should be used. A small quantity melted on to a silver probe forms an excellent weapon for fighting these apparently insignificant but very painful lesions. It is no uncommon experience to find a child taking its food again after a small erosion has been touched with this substance.

The larger ulcers, especially, are to be treated in this manner. Cysts must be opened, the sooner the better, by a free incision. If they should fill up again, cauterization of their walls should be resorted to. The treatment of those forms due to dentition will be discussed in the proper chapter.

II.

STOMATITIS APHTHOSA.

IN discussions on this subject we find confusion most dire. In looking through the literature it will be found that so many things are called aphthæ, and so many things have been called aphthæ, that but one of two courses remains to be taken,—either the term aphthæ must be discarded entirely, or we must make our definition of the term so precise that mistake is impossible. As has been pointed out before, the term comes from Hippocrates, and gradually it has been made to include nearly every affection of the mouth; thus, even modern books speak of it as synonymous with thrush or the ulcerative form. The first who gave us a definition for the modern acceptance is Billard ("Maladies des Enfants," p. 230 *et seq.*, Paris, 1837), where a complete discussion of the history of the subject may be found. Billard speaks of a stomatite folliculaire ou aphtes, but the adjective is to be construed more as referring to the form of eruption than to its location. To Bohn is due the credit ("Die Mundkrankheiten der Kinder," 1866) of having placed exact limits to our conception of what should be meant by aphthæ. It must be remembered, in this connection, that the accepted term has an entirely different significance from that which Hippocrates intended,—he, in all probability, had reference to the mycotic form only when he speaks of ἄφθαι. As a result, some modern authors still speak of aphthous sore mouth as thrush; but, unfortunately, whenever this is done a description is given which shows very clearly that the author is describing several forms under one heading which have no possible connection with each other. If all authors would unite and give to the term aphthous or aphthæ (either adjective or noun) the Hippocratic sense there certainly could

be no objection raised. But as it is, the two courses before mentioned are the only ones possible. As far as rejecting the term altogether is concerned, Bohn has done so much to establish the identity of the affection, and it has been taken up by so many authors (all the German, some of the French, English, and American), that it would seem like taking a retrograde step to drop the term. In addition, the confusion that already exists would be increased, and that which has been gained by precision would be lost. As a result the term stomatitis aphthosa has been retained to mean that form of disease described by Bohn.

Definition.—By aphthæ are meant spots, of different color, appearing within the mouth, situated under the epithelium, surrounded by an areola, again of varying color, which run a peculiar course during their existence. As far as the nature of these spots is concerned there still exists considerable discussion. There are principally two opinions expressed. The one, that we are dealing with a vesicular eruption; the other, that we are dealing with a solid exudation between the cutis and epithelium. The great objection urged against the former view by Bohn and his followers, that they have never seen any fluid within the spots, is, apparently, a very valid one. But if we take as simple a matter as herpes, it will be seen that if we were to judge this eruption by the presence or absence of fluid within the efflorescences we might, in a great many cases, be led to the conclusion that herpes is also a solid exudation between the cutis and the epithelium. If to this there is added the fact that all forms of skin-trouble do and must, of necessity, take upon themselves a different form within the mouth than upon the skin, a great deal of the force of Bohn's argument is lost. It certainly must be accepted as a fact that the epithelial layer is regenerated much more rapidly within the mouth than anywhere upon the surface of the body. This, taken together with the constant bathing with fluids, under pathological

conditions even greater than in health, and the great disparity that exists between the two views can be readily cleared away. When we come to compare the clinical history of some forms of herpes and of stomatitis aphthosa, it will be found that the view which makes both processes due to the same causes is, to say the least for it, very enticing. When, added to this, there is a series of carefully-conducted bacteriological investigations which give a negative result, as far as pathogenic organisms are concerned, we will have to think even more seriously of this view.

As far as locality of eruption is concerned there can be but one opinion. Aphthæ appear in parts of the mouth in which there are no follicles; therefore the eruption cannot be follicular in the sense that it is the result of some process which goes on within the muciparous glands. This, on the other hand, does not prevent our acknowledging the fact that an aphthous eruption may appear at the mouth of a follicle any more than our accepting the fact that an herpetic eruption may develop at the opening of a sebaceous follicle or sweat-gland. Yet no one would think of calling herpes a follicular eruption.

Again, concerning the term aphthous ulcer, to which Bohn objects so strenuously. This depends entirely upon what may be defined as an ulcer; if there is necessary both a disturbance of continuity and the appearance of pus we can certainly not speak of the existence of an aphthous ulcer, as it is exceedingly rare to find pus in appreciable quantity the result of aphthous stomatitis. If we go further and accept Billroth's view, that the appearances in the intestines in typhoid fever must not be considered as ulcers because they have a tendency to heal (it being absolutely indispensable for an ulcer not to have the tendency to get well), then we can certainly not speak of the epithelial sores made by aphthæ as ulcers. On the other hand, the local conditions referred to above must be again taken into consideration. We must also bear in mind that we

are dealing with a term which is used with more or less freedom by the profession, and although, theoretically, such a loss of substance as is produced by an aphtha is not an ulcer, yet, for all practical purposes, it must be considered as such. As will be seen farther on, the epithelial loss produces symptoms just as well marked and as intense in their nature as if pus were being formed, or as if there existed a tendency to spontaneous healing.

Etiology.—Concerning the etiology of this affection we are completely in the dark, as far as positive knowledge is concerned. A great many views have been expressed and a great many things have been brought into causal connection with stomatitis aphthosa, but as yet no lesion has been discovered beyond those which will be described and which are absolutely inconclusive. The cause must be sought for either in the mucous membrane itself or in structures remote from it. As to the mucous membrane, there are many causes which might produce an eruption upon it of the nature described before. By means of applying caustics it is possible to imitate the appearance and course of aphthæ in the mouth (Gerhardt, Bohn). I have seen burns in the mouth, produced in one instance by the head of a burning match, which it would have been impossible to differentiate from aphthæ. But such external causes can be disregarded, as the eruption appears without any apparent external cause. It is natural that lower forms of life should have been accused of causing this trouble; but I have had eight cases of stomatitis aphthosa examined into by two most competent observers, Drs. Cameron and Freeman, demonstrators of bacteriology in the Medical College of Ohio, Cincinnati, with an absolutely negative result, as, after the most careful search, including plate- and tube-culture, only pus-formers were found. These were found in two out of the eight cases, and must, therefore, be looked upon as accidental. It can therefore be conclusively accepted that there exists no

localized cause in the mucous membrane. But one structure or structures remote from the mucous membrane could be accused of producing aphthæ,—the nervous system. Bohn shows that the greatest number of cases occurs between the tenth and thirtieth month after birth. Because the teeth come through about this time, and because prorruption of a tooth is frequently accompanied or followed by aphthæ, he comes to the conclusion, which is not unwarranted, that the process of teething has something to do with stomatitis aphthosa. We find aphthæ associated with any number of diseases,—pneumonia, ague, gastro-intestinal catarrhs, the acute exanthemata, etc. If there exists any connection between teething, pneumonia, ague, etc., and stomatitis aphthosa, bacillary origin being positively excluded, it must be through the nervous system. This view has been expressed by Bärensprung, who thinks that some forms of herpes-facialis may be due to lesions in smaller ganglia, just as herpes zoster is due to lesions in the spinal ganglia. There are objections to the acceptance of this view, however, as the eruption is not localized anatomically, as in herpes zoster, and frequently it is too general to be explained by the affection of one or two nerves. That an eruption can be produced by affection of nerves or nerve-centres is a fact accepted by dermatologists (Kaposi, Sattler, etc.). The eruption thus caused is herpes, and when herpes appears in the mouth it is "stomatitis aphthosa." Bohn, who in the beginning of his excellent article insists on the non-existence of vesicles, at the end of his chapter compares aphthæ with eczema or impetigo. Anatomically speaking, eczema is a process characterized by serous exudation and impetigo by purulent exudation. No one could claim that aphthæ are characterized by either of these two products, and when eczema does appear upon a mucous membrane (of the nose) there is no difficulty in recognizing it as such and no hesitation in separating it from an aphthous eruption. The fact that aphthæ may be found in

children with impetigo is of no possible value as establishing any connection between them. If we grant that aphthæ come out in groups (which will be shown to be the case), and if we admit that a vesicle in the mouth would present all the characteristics of an aphthæ, we are forced to the following conclusion: aphthæ are eruptions characterized by vesicles which appear in groups. This, it will be seen, is an exact definition of herpes.

Occasionally cases are reported in which the evidence seems to point to the contagiousness of this form of affection. There is no doubt that two or more cases will sometimes happen in the same family. Careful inquiry will almost always result in establishing the fact that the aphthous process is produced in these cases by the same cause; that the aphthæ are due to a disease, endemic or epidemic, which has attacked the various members of a family. In some instances we may be left entirely in the dark concerning the nature of an apparent epidemic; but the fact must not be lost sight of, that stomatitis aphthosa is a conglomeration of symptoms the exact nature of which eludes discovery.

The attempt has been made to bring this disease into relation with the hoof-and-mouth disease of cattle. If this should be proved in every instance, hoof-and-mouth disease must be very much more common in cattle than we have any reason to suppose. The possibility of a connection cannot be denied, and, if proved, would place aphthæ among the infectious diseases. Certain it is, however, that the cases which have come under my observation and the eight cases examined, mentioned above, were not of this nature. Chyrim (*Jahrbch. f. Kinderheilkunde*, N. F., xxiii.) reports an outbreak of hoof-and-mouth disease among the cows of the Model Dairy at Frankfurt-am-Main. The attempt was made to determine whether drinking the milk from the diseased cows had the effect of producing aphthæ, but the results were unsatisfactory. Fifty-three physicians answered questions relative to their patients

who took milk from the dairy ; out of this number twelve noticed eruptive diseases in the patients. In eight of these no connection existed between the milk of the dairy and the eruption, as the patients took milk from other cows. In the remaining four one physician reports herpes of the upper lip and throat, another reports two cases of skin affection, another vesicles upon the mouth and lips, and a fourth two cases of stomatitis aphthosa. So that, after all, there are but two cases left, and the final conclusion of the author seems justified, "that those consumers who remained true to the dairy did not suffer," which is the same result arrived at in the epidemic of 1877.*

Symptomatology.—Setting aside whatever general disturbances may be concomitant with the disease upon which aphthæ are engrafted, the symptoms are principally confined to local manifestations. Preceding the eruption of aphthæ there is usually present more or less stomatitis catarrhalis. This may be due to the disease producing the stomatitis aphthosa (malaria, pneumonia, etc.), or may be produced by the aphthous process itself. We find an analogue in herpes zoster facialis when the gums or cheeks become affected, and redness is always present even if no distinct eruption appears.

The aphthæ appear with lightning rapidity. A mouth which has been examined and found slightly reddened will, the next day, have an extensive eruption of characteristic lesions. These consist of small subepithelial whitish or yellowish-white spots, appearing singly or in groups, which may develop in any part of the mouth. They are not unilateral and, probably, are not confined to the cavity of the mouth (they not infrequently extend into the pharynx). The eru-

* The author has taken pains to examine cows supplying milk to patients affected with stomatitis aphthosa, always with negative results. He has further been told by veterinary surgeons that few, if any, cases of foot-and-mouth disease have occurred among cows in this country.

tion as such is very short-lived,—after from twelve to thirty-six hours the epithelial covering is soaked off, and there is left the so-called aphthous ulcer. This is characterized by its outline, formed by a slight depression surrounded by a red margin (the latter also present in the former state), and its floor being lined by the original contents of the vesicle. Where two or more aphthæ have developed close enough to each other, we find the ulcer becoming serpiginous, in that two or more have run into each other. After a few days more the epithelial layer begins to be regenerated, the small mass at the bottom of the ulcer is enclosed by this layer encroaching upon it from all sides, it is lifted up and projects beyond the level of the mucous membrane, and finally disappears. Or the floor of the ulcer is cleared, the exudation being washed away, and there is left a surface denuded of epithelial cells, which will bleed only when rudely touched. Again, some aphthæ will be absorbed without the outer epithelial layer breaking. When there are complications (stomatitis ulcerosa) the aphthæ sometimes become infected, and then we have a true suppurative process going on. As a rule, the aphthæ appear in crops,—the one succeeding the other,—so that the course of the disease may become somewhat protracted,—ten to fourteen days. Cases lasting beyond this time are much rarer in children than in adults.

The exudation as it is found in the ulcer will be found to be made up of small, indifferent cells, some fibres, and several varieties of lower forms of life usually found in the mouth, but not pathogenic. All the cases examined into were free from pathogenic forms which could explain the occurrence of the eruption.

The denudation of the epithelial layer is covered up with new cells and no cicatrix is left, because the connective tissue is not affected. The young epithelial cells are at first opaque, so that a white spot is left where the aphtha was; in a short

time, however, this disappears unless the process was complicated by some other form, when a slight scar remains.

While this whole process is going on the subjective symptoms vary enormously. Some children are very little affected by stomatitis aphthosa; indeed, as a rule it is only the denudation and its contemporary irritation and reaction which produces symptoms. These are the same as described under stomatitis catarrhalis,—salivation, pain, restlessness, loss of appetite, etc. Bohn lays especial stress upon the fact that the saliva in stomatitis aphthosa is not fetid. This can be verified in every instance, unless a complication exists with stomatitis ulcerosa, which is not very rare. In some instances the eruption is so extensive that the whole mouth is covered with it and produces the picture of a diphtheritic inflammation. If differential diagnosis is not possible in the first instance, a day of waiting will clear up the whole picture, as by that time some of the spots will become denuded and symptoms of general infection will have appeared.

Prognosis.—This is absolutely good. The same that holds good for stomatitis catarrhalis is also true here. We are dealing with a self-limited disease, which does no harm except in that it may affect the general health of the patient. As far as the local trouble is concerned, in an otherwise healthy child, stomatitis aphthosa is to be looked upon as a painful but harmless affection. It is barely possible for the ulcers produced by this disease to become infected with other poisons (some cases reported by Schrakamp are possibly of this nature), but this is, fortunately, of rare occurrence. Good or bad general conditions of health seem to have very little to do with the frequency of the eruption,—it is very easy to say that rachitic, syphilitic, badly-nourished, etc., children are more liable to aphthæ than healthy ones. Beyond the fact that this form of trouble is concomitant with a great many acute diseases these statements are perfectly gratuitous and require to be

proven. A form of chronic ulcers seen in adults is very rare in children. These are catarrhal in nature, come and go, last for a long time, and are usually accompanied by general disturbances. It is a mistake, however, to call these ulcers aphthous, as they do not possess any of the characteristics of aphthæ, not the least important, for the latter, being their tendency to spontaneous healing. These chronic catarrhal ulcers have been confounded with aphthæ, and what is true for them has been ascribed to the aphthous process. For the explanation of their general constitutional effects we refer to the previous chapters. Relapses are not common in children after the affection is once healed, another evidence that the general condition has little to do with the appearance of this eruption. It will occur that in a reduced child the process does not have a tendency to get well,—just as an ulcer upon the skin under the same conditions would not heal. In such cases these ulcers, as has been indicated before, may give rise to a great deal of trouble.

Treatment.—The object of treatment is to give relief from pain and prevent infection. The former, and possibly the latter, is accomplished by touching each ulcer with nitrate of silver. The treatment is identical with that recommended for catarrhal ulcers and gives just as much relief. I have never had good results from cocaine, recommended by some authors in troubles of the mouth (Bockhardt, *Monatshefte f. Dermatol.*, v. ii., 1886), and would hesitate to employ the very strong solutions (ten to twenty per cent.) recommended. Baginsky speaks very highly of permanganate of potassium (0.10 to 15.00) and considers it almost a specific, curing the affection in a short time (*wenigen Tagen*). Chlorate of potassium is unnecessary, as much so as the great number of external remedies that have been vaunted and applied. The same rules for diet put down in the previous chapter also apply here. The fact must never be lost sight of that a pure, uncomplicated case of stomatitis

gets well of its own accord, and all the physician need do is to watch, give relief, and prevent any complications by hygienic measures.

BEDNAR'S APHTHÆ.

In 1850, Bednar's small but, clinically, very valuable book appeared, in which was described a peculiar form of lesion of the mouth, which has since been accepted as Bednar's aphthæ ("Die Krankheiten d. Neugebornen u. Säuglinge," etc. Vienna, 1850). He states that this form is only found in infants from the second day after birth to the age of six weeks. There are five different forms, characterized by the locality and nature of the eruption,—the first three are found upon the hard palate, the fourth upon the soft palate as well, and the fifth is hemorrhagic. They are preceded by an injection of the mucous membrane, and then follows an exudation, gray or yellowish-white, subepithelial. This breaks down and leaves an ulcer. They are found in the posterior portion of the hard palate either on one side (first form), symmetrical (second form), or combined with one upon the palatine suture, but always near the velum palati. Such, in brief, is Bednar's description. It is not difficult to see that a great many processes may run their course and give rise to symptoms akin to those described. Such is the case, and we find at least three different conditions, perhaps more, which it is impossible to distinguish the one from the other. There is that process which is found in the mouths of newly-born infants as well as upon their skins, the development of milia; when these ulcerate from one cause or another, they give rise to appearances similar to the aphthæ of Bednar. There are retention cysts, very small, like acne, which may also be followed by ulcerations (Bohn). Epstein claims that small defects, congenital, exist in the mucous membrane filled with epithelial detritus which simulate Bednar's aphthæ. The same author states that true ulcers, produced by decubitus, may occur upon a mucous membrane affected by catarrhal stomatitis caused

by nursing. For this he gives an anatomical explanation in that the part of the mucous membrane affected becomes most tense and anaemic during the act of nursing, and therefore more liable to be affected by pressure than any other part. Comparatively recently Fischl (*Prag. Med. Wochenschrift*, xi. 41, 1886) has made observations which throw some light upon the etiology of Bednar's aphthæ. He took a large number of children in the Foundling Asylum at Prague and divided them into three groups. In the first group the mouth was left alone, not washed nor cleansed in any way; in the second group the mouth was washed and cleansed regularly; and in the third no especial attention was paid to the matter, so that some were and some were not washed out. The result was that in the first group five per cent. were affected, in the second fifty-four per cent., and in the third fifteen per cent. The ulcerations of the soft palate were also noticed most frequently in the second group. It seems, then, that the most common cause for these aphthæ is violence; and the statement will certainly be borne out by the experience that, when this form of trouble is noticed at all, it is much more common in hospital than in private practice. In private practice the nurse is under the observation of the mother, in hospital practice she is apt to be too zealous in the performance of her duty; when she is ordered to keep the mouth of a patient clean, it is done hurriedly and, perhaps, not too gently. The ulcerations upon the velum are just in the locality where the end of the finger would touch when introduced into the mouth, and those upon the hard palate can be explained just as readily by the sweeping motion of the back of the finger. While it cannot be denied that these aphthæ may arise spontaneously in any of the ways indicated before, it must be confessed that the origin by violence must be looked upon as the most common.*

* Ulcers far forward, upon the hard palate, are not infrequently produced by the rubber nipple in artificial feeding.

Again, these ulcers are self-limited ; their tendency is to get well. The symptoms produced are those of pain in nursing only, and the consequences of not taking food. They are apt to be complicated by the development of thrush, and sometimes (as in two cases of Fischl, *loc. cit.*) may terminate fatally by producing gastro-intestinal disturbances when they persist for too great a length of time. The term is used as a clinical one, just as it was used by Bednar ; it represents a clinical picture, produced in different ways, and his description is just as true to-day as it was when he first published it.

Treatment.—Bednar says, “The disease cannot be shortened by any remedy, and in the absence of any dangerous complications its termination is always favorable ; therefore it is superfluous to paste the mouth with mucilago or to wound it with caustics.” The disease is rare in this country, but it does occur. In the cases that I have seen I have remembered Bednar’s injunction, and they have all recovered without any untoward symptoms. Those complications that may arise must be treated as such, but it is unwise to do more than is already being done in the effort to repair damage resultant upon various causes. The most common complication is stomatitis mycosa, which can be easily avoided and just as easily treated. The general disturbances, dyspepsia and intestinal lesions, must be overcome and the general nutrition of the infant must be watched.

III.

STOMATITIS MYCOSA.

Synonyms.—Thrush, Soor, Mundschwämchen, Muguet.

THE nature of this disease, now so clearly understood, was entirely unknown until the parasitic growth which causes it was discovered. On account of the fact having been thoroughly established, and because the life history of the parasite is comparatively well known, thrush becomes one of the diseases which can be looked upon as a paradigm by which other infectious diseases can be regulated.

The historical development of the subject may be divided into two periods,—that before the discovery of the cause of the disease (about 1840) and that following this date. In the first period we find the older writers, and especially the French authors. It is almost a certainty that Hippocrates described thrush under the heading of *στόματα ἀφθώδεα*, and Galen was also acquainted with the affection. The authors following them looked upon the affection either as ulcerative (Avicenna) or vesicular, papular or pustular (Boerhaave, Rosen v. Rosenstein). Rosen (German edition, translated and edited by Murray, professor in Göttingen, 1774) has, like all his predecessors and a great many of his successors, described many forms under the head of “Schwamngen.” He has evidently seen cases of diphtheria which he writes about, possibly some other forms of stomatitis, but, without doubt, cases of thrush. He has made accurate observations in connection with the latter,—about the effect of cleanliness, the possibility of producing irritation of the nipples of the nurse, a connection between gastro-intestinal troubles and the sore mouth,—and advises the use of some remedies which, it is strange

to say, are still favorites with some authors on children's diseases (rhubarb and magnesia!).

In 1786 the Société Royale de Médecine offered a prize of twelve hundred livres on the causes of the disease known as "millet, blanchet, muguet" (thrush). This was done because of the fact that so many children were dying of the affection at the Hôpital des Enfants. The prize was divided between four, out of six competing, one of whom, Van Wimperse, succeeded in localizing the affection anatomically. This was, as Bohn states, the first attempt to describe the disease as an independent affection, and the result was an impetus given to observation in a different direction from that of former authors. After 1826, when Bretonneau first described diphtheritis, a name which he afterwards changed to diphtheria, it was held by a great many French authors that thrush was diphtheritic in nature, and even to the present day we still find French writers speaking of a "stomatite pseudo-membraneuse" when the invasion of thrush is very extensive. From this time until the discovery of the cause of the disorder very little progress was made beyond the discovery that the disease did not limit itself to children, but was also found in adults suffering with lingering or wasting diseases. The result was that great stress was laid upon this fact, the local nature of the disease was overlooked, and the fearful mortality spoken of by Valleix ("Clinique des Maladies des Enfants nouveau-nés," 16, 1838)—twenty cases dying out of twenty-two—asccribed entirely to this affection. This view, somewhat remodelled, was again taken up by Parrot (1874), who ascribed the predisposing cause of thrush in all instances to the condition he calls athrepsia, a view which, it will be seen, is altogether untenable. We now come to the second historical period, in which the cause of thrush was first discovered. There are a great many observers who saw the mould, but to Berg, of Stockholm, is given the credit of first having observed it, at least of first

having described it accurately and making experiments with it, showing its nature, the possibility of cultivating it, and its inoculability. His description is the one to be found in most works and articles upon the subject of thrush; but Robin (1853, "Histoire Naturelle des Végétaux Parasites") first named the vegetable parasite "oïdium albicans," a name still employed, although subsequent observers have been unable to classify the growth under this heading. The old name is now chiefly used by French authors (Fossanagrives, Simon), who continue to quote the older experiments, although progress has been made since Robin. Grawitz (*Virchow's Archiv*, 1877, p. 546 *et seq.*), following the methods indicated by Brefeld, was the first to study the thrush fungus according to modern ideas, and with the following results: he obtained pure cultures in a fluid made up of a solution of glucose, one per cent. of ammonium tartrate, and mineral salts obtained by making an extract of cigar-ashes. He also used a decoction of baked plums or currant jelly diluted with equal parts of Pasteur's liquid. In these fluids he demonstrated that the thrush fungus could be cultivated, but only in a peculiar state,—that of spores with the mycelium badly developed; the more sugar there was present the greater the number of spores; the more salts, the greater the number of threads. From these he made pure cultures, and came to the conclusion that the yeast-cell or spore was the forerunner of the mycelium, and according to the nature of his fluid he could cultivate thrush fungus rich in mycelium or made up principally of spores which resembled yeast-cells. There are two ways, then, in which the fungus grows,—one from clusters of gonidia attached to the mycelium, another from free spores. He then states that the fungus of thrush is not oïdium albicans but the ordinary mycoderma vini, which produces a fermentation and which grows upon fruit juices, but only in the form of spores. Grawitz then furnishes the proof of his having described the thrush

fungus by taking a pure culture of the mycoderma vini and producing thrush in five young dogs which were fed upon cow's milk. About the same time Reess published his observations (quoted from Bohn), in which he comes to the conclusion that the thrush fungus is not an oïdium but a saccharomyces-producing fermentation. He was not able to convert the mycoderma vini into a thrush-producing fungus or *vice versa*, and therefore proposes the name *saccharomyces albicans* until the exact relation of mycoderma and the thrush-producer is positively settled. A. Baginsky (*Deutsche Med. Wochenschrift*, 1885, p. 866) has made some experiments by means of plate cultures on meat peptone, gelatin, and potatoes, which were considered pure cultures by Koch. On potatoes he obtained the yeast form, on bread the same, especially upon the surface, and very little mycelium. In test-tubes the surface proliferation was that of yeast-cells, while in the deep it was in the form of mycelium. He does not think the fungus is mycoderma, and mentions Stumpf (whose publication I could not obtain), who thinks that the fungus is a mixed one, made up of oïdium and yeast. Plaut (1887) completely disagrees with Grawitz, and claims that the plant is the *monilia candida*; he comes to the following conclusions: The plant does more harm in its mycelium form; it does not develop upon healthy mucous membrane; and, lastly, the best treatment is corrosive sublimate, applied in the strength of one part in a thousand of water. G. Roux and Linossier (1890) show why so much confusion exists, but up to the present the position of the fungus has not been accurately defined. According to these observers, pure cultures can be obtained in Esmarch tubes or by plate culture, and, in forty-eight hours at 15° to 20° C., before other colonies of mouth microbes have appeared, colonies develop that are made up entirely of yeast-cells. The enclosing membrane of the cells does not have the cellulose reaction; the protoplasm, at first, is hyaline and

homogeneous, but becomes vacuolated and has small mobile granules. Like other microbes, basic aniline dyes are taken up with great avidity, but the cells are not decolorized after using Gram's liquid. There is no nucleus, the nuclear substance being in a diffuse state throughout the protoplasm.

The yeast form is always produced upon neutral or slightly alkaline, peptonized gelatin, and this form can be confounded with any one of the *saccharomyces*. This is the mature form which is modified according to the food upon which the fungus develops. Budding goes on with very great activity, especially upon solid substances like carrots, apples, etc., producing torula forms, "veritable bouquets of yeast-cells," which, under favorable circumstances, increase in size. Some of these cells become filamentous and then all the forms between the yeast and the globulo-filamentous can be observed. The production of filaments goes on in two ways: one, by the production of a daughter cell, which becomes a filament; and the other, by the pushing off from the mother cell of a protoplasmic process which is separated only when the thread is thoroughly formed. The latter process can be mistaken for spore formation, but by careful observation this can be excluded, as both processes go on in the same specimen, and their comparative frequency depends upon the nature of the culture medium. Under all circumstances, we no longer have the right to call the globulo-filamentous form, mycelium, and the yeast form, conidian, as both of them form spores, and therefore both have a right to the appellation of mycelium. They are different aspects of the same cell, depending upon the culture medium, so that one culture may be made to produce all the intermediate forms.

Two distinct kinds of elements are produced by the budding of these threads; the yeast form and new threads. The latter do not take part in reproduction, but disappear rapidly when a new culture is made: no purely filamentous form has ever

been obtained, though cultures upon gelatin peptone with cane-sugar approach very near to this condition.

Cultures upon boiled carrots in Roux tubes are best for a study of the fungus. At first there is a tendency to the production of filaments, but after forty-eight hours yeast forms alone are found, which persist; the filaments being found only in the deeper layers in contact with the carrot. Everything else being equal, solid media are better for the development of the fungus than liquids.

No ascospores are formed; therefore, according to these observers, the fungus is not a *saccharomyces*; the chlamydospore, which is observed, has been seen in its various stages by others, but never been thoroughly appreciated: for the purpose of studying this Nägeli's fluid No. 1, with one to five per cent. of saccharose, is the best medium, and in cultures far removed from the original from the mouth. These spores differ from all other portions of the fungus in their micro-chemical reactions as well as in their appearance. They react differently to methylene blue, osmic acid, and eosin. When they first make their appearance their contours are well rounded, their protoplasm is less hyaline than that of the conidia, and their membrane is thicker. They attain their maximum growth in forty-eight hours, and are three to four times as large as the yeast forms; they are spheres with a very thick lamellated tunic, enclosing granular, punctate, and proliferating protoplasm. The cells to which these spores are attached have been called preterminal by the author; the three or four nearest to the spore contain glycogen; the spores themselves show, with the iodine reaction, alternate layers of brown and yellow, which, during development, disappear to give way to a uniform reddish tint.

The chlamydospore contains a central body which can be forced out of the capsule by pressure; this is made up of granules possessing Brownian movements, arranged around a

larger body, the latter not taking up ordinary coloring matter. During life it was found that the granules began to disappear, the body around which they are arranged to increase in size, and to become surrounded by a membrane; coincident there is a complete disappearance of glycogen and protoplasm in these preterminal cells. The results of these biological researches are that thrush is propagated in three ways: (1) in the filament formed by conidia; (2) in the yeast form, by isolated conidia; and (3) by spores. It cannot be proven by direct observation that the chlamydospore takes part in propagation, but on account of its presence the fungus is not a saccharomyces nor is it monilia candida. Cells of involution, pseudosporangia, are also found, which may give rise to confusion.

In regard to pabulum, the authors state that the more complicated the molecular structure of the culture-liquid becomes the more complex the forms of the mould. Cultures do not always behave in the same way under the same conditions. The reaction is of most importance to us as physicians; slight acidity has no influence upon the growth of the mould; when the dose is sufficiently great to put obstacles in the way of growth, the filamentous form is produced. Moderate alkalinity tends to keep up the yeast form; great alkalinity has toxic properties. The authors point out that alkaline treatment is beneficial in three directions; it is possible to destroy the fungus with alkalies; the yeast form, produced in an alkaline reaction, is much easier of removal than the filamentous, and, lastly, the fungus cannot thrive upon milk unless the milk-sugar be converted into lactose by the saliva; alkalies will prevent this change from taking place, therefore no carbohydrates food will be present for the mould.

As a final conclusion, after having examined into the value of a great many articles as food for the fungus, the authors state that its alimentary requirements are distinctly different from the yeast of beer.

Until all discussion ceases relative to the exact position of the thrush fungus, we will use the term saccharomyees, principally because it is a compromise term and because it shows positively that we do not believe in the existence of oödium as the cause of this disease.

Etiology.—There can be no doubt of the fact that the saccharomyces is the prime cause of the stomato-mycosis. But, as is the case with so many infecting substances, it is necessary that the fungus be deposited upon soil which is favorable for its growth before a diseased condition can be produced. We will have to examine as the two etiological factors, first, the fungus, second, the patient upon whom the fungus grows. The natural history of the fungus is, briefly, as follows: It is found pretty widely distributed; in the human being, upon every mucous membrane,—the respiratory, the alimentary, the genito-urinary,—and, in several instances, in the parenchyma of the internal organs, the brain (Zenker), the lungs (Parrot, Bireh-Hirschfeld). E. Wagner discovered the fungus growing into blood-vessels, and from thence the possibility of a general infection is a matter readily explained. In the wards of hospitals where the disease is most common the air will probably be found full of spores, which develop as soon as they come in contact with the proper soil. On account of the fact that most of the observations which have been recorded have been made as the result of hospital experience, they should be taken with some allowance, for the air being loaded with these germs, it is impossible to draw conclusions to which some objection could not be raised. A single observation made upon an infant in a private family under good sanitary surroundings would, therefore, be of more value than those made in wards where the poison is ever present. Unfortunately, however, we come here to an insurmountable difficulty. If the germ is the myeoderma vini, it is ubiquitous, and we could hardly determine where it came from in the individual case, except from the air. All authors agree that the disease is found most com-

monly in infants during the first two or three weeks of life, although it may be found at any age. Several observers (Trousseau, Haussman) have found the fungus upon and within the female genitals, and Haussman ("Die Parasiten d. weibl. Geschlechtsorgane," Berl., 1870) lays stress upon the fact that infection of the newly-born takes place from the genitals of the mother during birth. The possibility of such an infection cannot be denied, but no proof of the fact has, as yet, been offered. Thrush of the vulva or vagina is rare (the large works on obstetrics and gynæcology do not speak of it at all); but admitting that the parasite does occur without symptoms, the proof would have to be furnished that children born from such mothers are more liable to stomatitis mycosa than others. Since my attention has been especially called to a possible causal connection between the two conditions, I have examined pregnant women coming under my care for the last four years in this direction. During this time I have found but two cases in which thrush of the vulva could be diagnosed,—one a diabetic patient, the other suffering from vulvitis with lacerated perineum and prolapse of the posterior wall of the vagina. In neither of these cases did the children show signs of thrush, although the child of the first mother had to be brought up without mother's milk, and in neither instance were efforts made to prevent the development of the parasite if it had been present. Every one who has studied the subject carefully will have come to the conclusion that thrush can be carried by the nipple, either of a nurse or of the feeding-bottle. The latter is especially the case in hospitals, when the nurses are not too careful as far as cleanliness is concerned. For a short time I was officially connected with a foundling hospital, principally for the purpose of helping in an attempt to reduce the fearful mortality which existed in the institution. I had the infants taken to a different building; unfortunately, I had no control over the nurses, so that I found myself thwarted and gave up in despair. Of some twenty infants

brought in, every one had thrush ; as far as I could discover, only one of the patients survived after having been removed from my care. The nurses prepared a large quantity of food, filled three or four feeding-bottles of the patented variety, and these were passed from one child to another. The bottles were never emptied, nor, as far as I could find out, ever cleaned. If we were to judge of the nature of stomato-mycosis from this experience, what an unsatisfactory condition we should find ! yet this has been done, especially by the older French writers, and even to-day the same thing is being done.

I have seen several instances in which apparently perfectly healthy infants have been affected with thrush. Epstein (*Prag. Med. Wochenschrift*, 1880) mentions the case of a woman who nursed two children, one of whom had thrush and the other one did not get it. I have met with the same experience, but one which renders conclusions difficult to be drawn. A woman presents herself with her infant, apparently healthy in every respect (details are unnecessary) except that the child has thrush. In the same ward there is an infant with cholera infantum,—bottle-fed, marantic ; in order to save this child's life the mother of the infant with thrush is utilized as nurse ; the child recovers without thrush. Here is a case in which a healthy child has thrush and a sick child who is exposed to infection does not get it. As far as general good health is concerned, it must be admitted, then, that when it has an effect upon the production of thrush it must be an indirect one. That such is the case must be admitted upon close examination ; the indirect effect is produced by some change in the mouth by means of which a proper soil is formed for the fungus. In what does this change consist ? A great many theories have been advanced in solution of this fact, which has been known for a long time.

It has been stated that for the development of thrush flat or squamous epithelium is necessary. At present there are so many cases on record in which the *saccharomyces albi-*

cans has been found in places containing no flat epithelium (stomach, small intestines, lungs, brain, blood-vessels, etc.) that this cannot be admitted as an etiological factor. Where Grawitz has found the yeast-form cell only in the stomach, Parrot ("L'Athrepsie," p. 224) claims that both mycelium and spores are found superficially, which his plates do not show. This latter fact, however, is not important in this connection, as we wish to show only that flat epithelium plays a very secondary rôle in the production of thrush. There can be no doubt but that it is observed most frequently in the mouth and the pharynx, but this does not mean that it does not exist in other places.

The only etiological factor which is admitted on all hands is the existence of a stomatitis catarrhalis, either before or with the appearance of thrush. A child suffering from any form of stomatitis (as has been mentioned in connection with stomatitis aphthosa) is more liable to thrush than one without such an affection. Whether the catarrhal stomatitis is essential to the production of thrush, or whether another element is to be taken into consideration, is difficult of decision. Rajewsky has proven that an irritation of a mucous membrane is necessary before it can be made diphtheritic. Is it the irritation or the disturbance of continuity of the epithelial covering which makes the mucous membrane pervious to the poison? In a case of thrush, is it the mechanical dislocation of the swollen epithelium, the separation of the cells,—all concomitant with stomatitis catarrhalis,—that predisposes such a membrane to thrush? There are some facts that point in this direction. Every one who has studied the subject admits that spores of the saccharomyces are found in the mouths of perfectly-healthy children: in cultures made for me they were found four times out of twenty-two. They do not seem to develop under these circumstances; they do not obtain a foothold; they are, in all probability, prevented from developing by the movements within the mouth, especially in older children. Given a case,

however, in which the mouth, especially of the young infant, is slightly bruised or its epithelial coating injured from attempts at nursing from badly-formed nipples, from a hard nipple of a feeding-bottle, with a cleft palate or what not, and thrush follows very rapidly. It will be seen from this that the feeding-bottle may be deleterious in more than one direction as far as thrush is concerned. These facts, taken in connection with some observations in the pathological anatomy of thrush, would make it seem that the results of a catarrhal trouble are to be feared more than the catarrhal stomatitis itself; in other words, that a mechanical condition must be produced which is favorable to the development of the parasite and which can exist either with or without stomatitis catarrhalis. It is impossible to conceive of an erythema or inflammation of the mouth which does not produce conditions favorable to stoma-mycosis, and all modern observers admit the intimate connection between these two conditions. Resulting from this comes the statement that all those conditions which produce stomatitis catarrhalis will favor the development of stomatitis mycosa.

It is a self-evident proposition that when the parasite is where circumstances are most favorable it will grow best. For this there is necessary an amount of comparative rest which can only be obtained under certain conditions. From the pathological anatomy it will be seen that it grows in places where it is least disturbed. From the knowledge obtained through clinical evidence we know that it grows best in those subjects who subject their tongues or their mouths to least motion. We find it, therefore, principally in infants, or in children sick with other diseases; in adults, in all forms of wasting disease or in acute disease accompanied with great debility,—all of which presuppose a condition in which the function of motion of the upper part of the alimentary tract is greatly diminished.

Pathological anatomy.—The parasite is taken up between the epithelial cells, so that at first the surface of the mucous membrane is comparatively free from any eruption. As a rule, the first development takes place so as to separate one layer of epithelial cells from the other ; this development is in the form of spores without mycelium. From this original implantation the parasite grows in both directions,—towards the surface of the mucous membrane as well as towards the connective tissue. In either direction do we find mycelium being developed,—to a very limited extent in the direction of the free surface, but thoroughly well in the direction of the connective tissue. Once the basement mucous membrane has been perforated, and the character of the growth seems to change so as to produce those pictures which have been put down in the books as the classical appearance of the parasite. From this method of development it will be seen why a squamous epithelial coating will favor the growth of *saccharomyces albicans*, and why, on the other hand, mucous membrane lined by cylindrical epithelium is not favorable to its growth.

In the mucous membrane with flat epithelial cells, the parasite can develop between the individual layers of cells ; in the mucous membrane with cylindrical epithelium, there are no layers between which the spores can develop. When they fill up the follicles then the growth goes on,—it is the surface growth that we are referring to,—but especially well into the submucosa and the nervea. Very much has been said about the exact relation which is borne by the parasitic growth to the outer epithelial layer of the mucous membrane. A careful investigation of each case will show that the beginning of each growth is usually as has been described ; that the *saccharomyces* then develops so as to implicate all the various layers of the epithelium. In attacking the most external layer it develops between the cells, raises them up, surrounds them, embeds them within its rapidly-increasing growth, so that, finally, it is im-

possible to distinguish epithelium from parasite unless the microscope is used. These facts are of great importance from a therapeutic stand-point. The implication of blood-vessels, which Wagner affirms and which Parrot denies, is a question which does not interest us for the present. But the affection of surrounding tissue as a result of the presence of the parasite is of some importance. The vegetable produces all the signs of irritative change,—proliferation of the cells, especially their nuclei, but no pus. The evidences of irritation are of the most transitory nature and vanish very quickly when the parasite is removed. The question whether or no pus is formed is at the present day of no importance whatsoever. It must be taken for granted that the *saccharomyces albicans* does not belong to that class of parasites called the pus-formers, as the formation of pus must be looked upon as the exception and not the rule. The extension of the parasitic growth to other parts of the body has already been referred to and will receive discussion in connection with the symptomatology.

On account of the nature of its inception, the growth begins in the form of small spots, which may or may not become confluent. It may then be propagated either from this first crop or, what is more likely, in mild cases, two or more places may become inoculated from the same source. In microscopic preparations we sometimes see one islet connected with the other by threads of mycelium in the connective tissue. In violent cases a deposit of a mass occurs, leaving very little healthy tissue.

Symptomatology.—It has been admitted that a child, perfectly healthy in all respects, can be infected with thrush, and the attempt has been made to show that the first lodgement of the parasite is due more to mechanical causes than to any other circumstance. Yet the former proposition must be accepted as the exception and not the rule, and the latter as signifying that children whose mouths are otherwise affected are, as a rule, more liable to these mechanical conditions than healthy ones.

It will follow, therefore, that the symptoms of stomatitis mycosa are of a complex nature; those due to the stomatitis and those due to remote conditions, either predisposing, coexisting, or following the lesion of the mouth.

It will be seen that the term stomatitis mycosa has been used to designate the affection under discussion. This has been done because, in every instance, there are present the evidences of a stomatitis which is due to the irritation produced by the fungus; therefore a stomatitis mycosa. The subjective symptoms produced by the fungus, in a purely local case of thrush, vary with the intensity of the affection. In some cases, when the affection is but slightly developed, the patient suffers very little, if at all. It can be put down as a rule that pain is present only when the corium is attacked. The mechanical disturbances produced may be varied and various, depending entirely upon the part of the mouth affected. In the beginning we usually see the tip of the tongue the seat of the trouble. With this the lips are affected, and from these two places the parasite may grow in all directions. Most commonly, the tongue suffers most; from it infection may take place upon the tonsils, and then we have the symptoms of an amygdalitis, difficulty in swallowing, painful swallowing, and, finally, absolute refusal of food. When the tongue and the lips alone are affected, provided always the fungus has grown into the corium, we get the symptoms described under catarrhal stomatitis. In those cases in which the saccharomyces has grown upon the oesophagus the symptoms may become still more intense. Cases are on record in which the whole of the oesophagus has been filled up with a cylindrical cast made up entirely of the spores and mycelium of the parasite. There is no doubt that the observations of the French authors (Valleix, Seux) are correct as to the frequency of thrush in the oesophagus. These observers found it in thirty-two cases out of forty-two which were examined post-mortem. Although

this ratio overestimates the comparative frequency of thrush of the œsophagus, yet we have no positive proof that it does not exist during life. It is certain that autopsies, carefully conducted, will show the presence of thrush in the œsophagus in a far greater number of cases than we have reason to suppose when judging from the symptoms alone. When a plug is filling out the œsophagus, swallowing becomes impossible; but, fortunately, the attempt is sometimes followed by vomiting, by means of which the plug may be expelled. The question whether the saccharomyces produces gastro-intestinal troubles has been answered in various ways. The French authors claim that it does, and, in addition, that intestinal troubles are almost a *conditio sine qua non* of stomatitis mycosa. On the other hand, Bohn and most of the German authors claim that the disturbances of the intestinal tract frequently precede the stomatitis, and can, therefore, not be looked upon as sequelæ. It has already been pointed out that intestinal troubles need not accompany, precede, or follow thrush, and it has been stated that hospital patients are not the class of subjects upon which observations of this nature should be made. Especially is this the case when the hospitals in which these studies were made are themselves taken into consideration. If the experience of private practice be considered, it will be seen that bowel troubles with stomatitis mycosa are the exception and not the rule. This is especially applicable to that better class of patients that watches its children intelligently. For when thrush is treated properly in its beginning, intestinal or gastric troubles are simply out of the question. On the other hand, disturbances of the gastro-intestinal tract are the rule when stomatitis mycosa is under full headway. It is probable that the saccharomyces is alone sufficient to account for attacks of dyspepsia when swallowed in great quantity; but it is certain that the taking into the stomach of great quantities of saliva, holding in solution the

chemical results of the biological activity of the fungus, frequently causes catarrhal troubles of the gastro-intestinal mucous membrane. When the fungus develops in the mucous membrane it produces the symptoms of well-marked disturbance. Statistics are wanting concerning the frequency of all these occurrences. But it will be seen that thrush as a slight localized affection and under proper conditions need not affect the patient very seriously, while an extension to the tonsils, the oesophagus, the stomach, or even an extensive localized invasion of the mouth, must always be looked upon as a serious matter because of the digestive troubles which may follow. Again, thrush, when developing in a debilitated patient, the debility due to any cause immaterial, whether from gastro-intestinal disturbances, typhoid fever, pneumonia, phthisis, or what not, becomes a very much more serious disease than in a healthy child. Thrush occurs in debilitated subjects, as has been pointed out, and the most common cause for debility in infants is disease of the mucous membrane of the gastro-intestinal tract; it was therefore quite natural that the two conditions should have been looked upon as bearing the relation of cause and effect to each other. The fact remains that the former simply bears the relation of predisposing cause to the latter, the saccharomyces being the real cause.

Formerly great stress was laid upon the appearance of intertrigo with thrush, and it cannot be denied that intertrigo, or eczema ad natem, does occur very frequently in patients with stomatitis mycosa. The explanation is to be found in the fact that infants who have disturbances of the gastro-intestinal tract frequently have intertrigo; but this is due not to the saccharomyces but to the chemically-altered stool which irritates the skin over which it passes.

We find the characteristic lesions of thrush in the mouth. The beginning, as has been stated, is most commonly at the tip of the tongue, and we here see small, discrete, grayish-white

spots. When these are carefully examined by reflected light, it will be observed that they are covered by epithelium and are surrounded by a narrow ring of injected blood-vessels. Upon attempting to remove them it will be found that considerable violence is required, and when it is accomplished there is left a red surface, slightly depressed, which bleeds very readily. The latter condition obtains for all the various stages of the eruption, unless the whole mucous membrane is very much swollen, when a slight depression cannot be noticed. In the next period of development the spots will have grown, not so much in diameter as in height, and it will then be seen that they project somewhat beyond the level of the mucous membrane. This occurs in a comparatively short time, and after it more or less general infection of the mouth takes place. The latter does not follow as a necessity, but if these first two states go on unnoticed, the chances are very much in favor of more or less general infection. After this the spots enlarge, sometimes meet, and then the whole tongue may look as if covered by a membrane, the color of which depends upon the color of the food. When not colored by the food the membrane looks a dirty grayish white.

Sometimes the eruption begins upon the lips, the cheeks, or the soft palate; as a rule, that part lying directly opposite to the place first infected becomes affected next. When thrush begins upon the tip of the tongue it is the mucous membrane of the lower lip which becomes affected; when upon the cheek it is that part of the tongue which rests against the infected cheek, so that a direct connection between the primary and secondary invasions can be traced out. Again, under such conditions, it will be found that the two eruptions are in different states. The difference does not exist where cases are very far advanced and the various spots look alike. The mucous membrane between the spots is usually very much injected, of a dark-red color, and showing evidences of catarrhal

stomatitis. At times the fungus drops off or is detached, and slight ulcerations remain which may again be filled up with the parasitic mass in a very short time, or may remain as ulcerations, rather intractable, and of a very chronic nature if left to themselves. These ulcers may be the source of infection from poisons of a different nature, and ought, therefore, to demand the attention of the physician.

The differential diagnosis has been left for consideration until the various forms of stomatitis shall have been discussed. There is one point to which especial attention must be called in this connection. The beginner is sometimes at a loss to decide whether he is dealing with small masses of coagulated milk which have remained upon the mucous membrane or with thrush. If a camel's-hair brush or the finger be applied to coagula, it will be seen that they can be removed without any difficulty; with thrush, difficulty will be experienced and there will be left the raw surface. When the appearances are studied with care it rarely becomes necessary to use the microscope for making the diagnosis positive. But where there is any doubt, the microscopical appearance of the saccharomyces will be found so positively clear that there can be no hesitancy in their recognition by the veriest tyro.

Prognosis depends more upon the patient in whom thrush develops than upon the thrush itself. A local process in an otherwise healthy child is perfectly harmless, especially when properly treated. Thrush in a debilitated, enfeebled infant may be the cause of death,—the straw that breaks the camel's back. Again, stomatitis in a child with bad hereditary tendencies may become a very serious affection. Furthermore, the place of development must be taken into consideration. A serious invasion of thrush in the œsophagus will almost always be fatal; one may not be able to diagnosticate its existence, and even when this is done its removal is next to impossible. The younger the child, the more extensive the eruption, the worse the prognosis.

Last comes the factor of treatment. Careful management will do most to lessen the mortality from stomatitis mycosa. This should be especially taken into consideration by hospital physicians. There is no possible excuse for the high mortality reported from thrush ; with the light thrown upon the subject from the laws of disinfection, cases can certainly be isolated without difficulty, so that the weak in the wards can be protected. In private practice it is a matter of extreme rarity to see a patient die from stomatitis mycosa, although all cases should be carefully treated, as many complications can be prevented which, although not directly fatal, may finally influence the child's condition of health.

Treatment.—Prophylaxis is of as much importance in this affection as the treatment proper ; but cleanliness is absolutely imperative in both. As to prophylaxis, it is necessary to remember that all slight abrasions of the mucous membrane may become infected with the *saccharomyces albicans*. Furthermore, everything must be watched which might, by any possibility, be a place for the development of the fungus in appreciable quantity. For this reason it is well to teach the mother or the nurse how to keep the nipples clean, and how to cleanse the mouth of the infant. Wet-nurses should always be carefully inspected, their nipples and the mouths of their children inspected before permission is given to nurse the child for whom they are engaged. When the child is brought up on artificial food, the whole apparatus for feeding must be kept scrupulously clean, and the attendants must be taught how to do this. Not only is this important as far as thrush is concerned, but also in a great many other directions. The best and, upon the whole, the safest disinfectant for the feeding utensils is exposure to the temperature of boiling water for a little while. But every part of the apparatus should be so arranged that boiling water can gain access to it, and that any deposit can be removed mechanically. When this is

rigidly carried out, infection becomes impossible even in hospitals.

When the diagnosis has been made the treatment proper will consist of two distinct parts: the first, the mechanical removal of the fungus; the second, its destruction. A moderate amount of violence is necessary to accomplish the first, and, in order to insure the carrying out of instructions, it is best to reduce instructions to a method. The attendants must be told to wash out the mouth at stated times,—for instance, between the times of nursing and immediately after nursing. It has been found that removal of the growth is easier when an alkali is used; for this purpose the sodium bicarbonate (one dram to a tumbler of water) is very serviceable. Whether or no it has antimycotic effects, as far as the *saccharomyces* is concerned, is debatable ground. In former days the assumption that the fungus could not exist upon an alkaline soil was taken for granted, and because the *saccharomyces* was followed by an acid reaction, therefore an alkaline remedy was the proper one. Even if the soda has no especial effect upon the parasite, it has its indications in thrush, not the least important being that it causes the epithelial covering to be removed more readily, so that we can get at the fungus; where the epithelial coating has already been removed, it causes the mycelium to be less adherent, solving mucus and the substance holding the threads together. In addition, the remedies to be used must be applied frequently—four or five times daily—and with a brush.

In using remedies for thrush, it has been my custom for years to avoid prescribing syrups; the orthodox borax and honey mixture has always seemed to me to add fuel to the fire. Any number of medicines have been recommended in the treatment of this affection: potassium chlorate, potassium permanganate, borax, boric acid, the hyposulphites, salol, etc. This fact alone shows that they are of secondary importance, for all have supporters, and all have been followed by good

results. If the physician but adheres to the mechanical removal of the fungus masses, cure will follow. Up to the present the remedy or remedies which will prevent the growth of the *saccharomyces albicans* has not been experimentally determined upon. As far as my own experience is concerned, I have rarely found it necessary to use anything but sodium bicarbonate. Occasionally, when ulcers are produced, it becomes necessary to touch them with silver nitrate, but in uncomplicated cases this is exceedingly rare. There are some cases which will resist any method or all methods of treatment. But no case, when taken in the beginning, should be allowed to spread; a careful examination of the mouth will reveal the points of development of the fungus, and their removal ends the disease as far as those places are concerned.

Calomel in small doses or corrosive sublimate very much diluted almost always act as specifics in intestinal troubles which are due to thrush. But the relation between intestinal troubles and thrush must always be kept in mind, and the indiscriminate use of cathartic alkalies or other laxatives must be prevented as doing the patient more harm than good, reducing his strength and being absolutely harmful and needless. Baginsky claims good results from resorcin, and warns against the use of too large a dose (from one-half to one-per-cent. solution—never more than one teaspoonful every two hours). It is difficult to conceive how this, or any other remedy, is going to produce an effect upon an oesophagus stopped up completely by plugs of parasitic growth. When a conjectural diagnosis of oesophageal thrush has been made, it seems most expedient to introduce the soft catheter into the oesophagus. In one case I have succeeded in gradually working my way into the stomach with a catheter; some of the masses were pushed into the stomach and were then removed by vomiting. The patient, however, died a few days afterwards, and post-mortem examination showed the oesophagus again filled up.

IV.

STOMATITIS ULCEROSA.

Synonymes.—French, Stomatite Ulcero-membraneuse; German, Stomacace, Mundfäule.

WE have to deal here with a disease with a very limited literature, with a most distinctive clinical picture, and one whose causation is as yet unknown, except as a matter of theory. To this might be added, a disease whose treatment is thoroughly well understood.

The historical development is that of comparatively recent times. We find the French authors first describing the affection either as a gangrenous process (Taupin, 1839), diphtheria of the mouth (Bretonneau, Troussseau), and, finally, as an ulcerative-membranous process (Barthez and Rilliet, and those following). In England, West was one of the first to publish an excellent description of the disease, and then to give us the remedy, which is almost a specific. In Germany, writers like Jörg, Wendt, Schnitzer, and Wolf (1826–1844) give descriptions of stomacace, which, however, are not always perfectly clear as far as our present knowledge goes, so that the credit of the first sharply-cut description belongs to Bohn (1866). From this time little, if anything, has been added to our knowledge upon the subject; all the hand-books contain more or less lengthy chapters upon this disease, with descriptions more or less accurate. The position of the disease in nosology, then, is well recognized, and all modern writers, notwithstanding omissions in description, seem to have seen the same thing when they write about ulcerative stomatitis. The various views that have been held concerning the nature of the disease can be omitted as subjects of historical importance, but as valueless at the present day.

Stomatitis ulcerosa is a disease characterized by a peculiar pathological process, which terminates in molecular destruction of tissue. It begins on the gums around the teeth, it never extends beyond the cavity of the mouth, and it has the power of inoculating other parts of the mucous membrane. It may be well to emphasize the fact that stomatitis ulcerosa does not occur where there are no teeth.

Etiology.—Much has been written concerning the cause or causes of this disease, but as yet we only have clinical evidence, which shows that in the majority of cases there are two factors at work,—the one general, the other local. It has been conceded by all authors that there are certain poisons which will produce a clinical picture identical with that of stomatitis ulcerosa. First and foremost comes mercury, then lead, phosphorus, and copper, to which might be added iodine. In these days, when we have almost returned to the mercury-therapy of our forefathers, it is well to remember that mercury will produce stomatitis, and much more rapidly in children than in adults. Indeed, fifteen or twenty years ago mercury was used with the greatest care in children, because of the knowledge of this fact, which seems to have been forgotten in our enthusiasm for antiseptic remedies; and not a little of the success of some physicians with medical idiosyncrasies was due to their not using mercury.

If we admit the mercurial stomatitis as typical of and identical with the stomatitis ulcerosa, it is possible to arrive at some conclusion regarding the nature of the affection. Mercury is partially excreted by the saliva, and accompanying this process there is more or less inflammation of the mouth. It is a notorious fact that where there already exists an irritation of the mucous membrane, in the form of a carious tooth, or the hyperæmia of alcoholics or of smokers, there the inflammation will take place with most intensity, and is frequently followed by the production of ulcers. If we examine into the process

as it is going on here, we are forced to the conclusion that we are dealing with a process purely local in its nature. This is quite true, for in many instances a mercurial stomatitis is produced long before systemic reaction has taken place, on account of prolonged administration of mercury. Yet the local effect upon the mouth comes from the general system, and the mercury is to be looked upon as predisposing cause as much as the immediate cause. In other words, to produce a stomatitis ulcerosa it is necessary that the mucous membrane be prepared in some way, so that the process itself can be continued. Before, we have stated that the mercury acts both as predisposing and immediate cause. The latter cannot be verified, except in that mercury will, in most instances, produce stomatitis ulcerosa when pushed far enough.

Naturally, the question of the *rôle* that is played by lower organisms would come up here as well as in every inflammation. In the investigations that I have made, the result was positive only in so far that the various pus-producers were found, which could have been expected. The assumption that the mercury causes the mucous membrane to be changed in such a way as to become a good soil for the development of these pus-producers could not be maintained. For it is not an ordinary pus-producing process that we are dealing with, as will be seen from the pathological anatomy, but one that is almost unique in its way. That there is some specific cause at work must be taken for granted, on account of the peculiar nature of the process, and that this cause is in the nature of a lower form of life, or some infectious agent, is proven by therapeutic measures. We know that stomatitis mercurialis can be almost indefinitely prevented by absolute cleanliness. We know, furthermore, that certain agents, having for their physiological effect the giving off of oxygen, will relieve and cure stomatitis ulcerosa most rapidly. We are, then, forced to the conclusion

that in stomatitis mercurialis, or ulcerosa, there is, first, a general cause (better systemic) and a local cause. The local cause in stomatitis mercurialis cannot be definitely ascertained, but reduces itself either to mechanical irritation produced by excreted mercury (lead, phosphorus, iodine, etc.) or some infectious agent.

If we now apply this knowledge to stomatitis ulcerosa in subjects not under the physiological manifestations of these remedies, it will be seen that clinical facts will give us data more or less satisfactory. As to general causes, Barthez and Rilliet say, "Il n'est pas une des maladies de l'enfance dans le cours desquelles elle ne puisse survenir" ("There is not a single disease of infancy during the course of which it could not develop." Barthez and Rilliet, vol. i. p. 201). With us there are certain diseases which are accompanied by this form of trouble more frequently than others,—the eruptive diseases, especially measles and scarlatina, malarial troubles, typhoid fever, pneumonia, and whooping-cough. Children affected with rachitis, syphilis, or tuberculosis are apt to have this trouble. Again, on the other hand, there are those children who seem to be comparatively healthy, in whom the least disturbance will bring on an attack of stomatitis ulcerosa. Cases will come under observation in which there will be repeated attacks of this disease, provoked by a bronchitis, a slight gastric disturbance, an attack of coryza. I have under my charge a child, now five years of age, who, since the appearance of his teeth, has had stomatitis ulcerosa follow almost every illness he has had, whether slight or severe. Except for a slight enlargement of the glands in the neck this child seems to be perfectly healthy.

Nearly all writers have laid stress upon the external surroundings of the patient as cause. Barthez and Rilliet (*loc. cit.*) state that the disease is endemic in some wards of some hospitals. Nearly all authors (Taupin, Bohn, Henoch) claim

an effect from damp, poorly-ventilated houses. Unsalubrious climate is also accused of causing this disease,—*i.e.*, rapid changes from warm to cold, from dry to moist, etc. The diet of a child must also be looked upon as causative. A poorly, badly-nourished child will be more apt to have the affection than one correctly fed, so that poor children are more liable to the disease than the children of well-to-do parents. Scorbustus has also been put down as one of the general diseases producing stomatitis ulcerosa. This disease is so very rare in children in this country that practically the relation is unimportant.

For local causes in the mouth we must look to the teeth principally. Bohn says, "Without teeth no ulcerative stomatitis." The explanation for this fact is to be found, probably, in that the gums form a favorable place for the poison to develop. That disease of adults known as dental pyorrhœa—shrinking of the gums—is frequently produced by the accumulation of tartar at the bottom of the teeth. Sometimes this form of trouble is nothing more or less than a true stomatitis ulcerosa, even in the adult. Now, while it is extremely rare for children to have tartar upon their teeth,—*i.e.*, during the period of first, or the beginning of second, dentition,—the production of this deposit shows how easily substances may accumulate upon the teeth around the gums. When we take into consideration that adults sometimes, even with the greatest care and cleanliness, cannot prevent this deposit of tartar, it seems very rational to believe that children whose mouths are apt to be imperfectly cleaned, if at all, may have substances deposited upon their teeth. Now, given a child which has its gums prepared by some general trouble for the reception and growth of the poison or irritant of stomatitis ulcerosa, and the origin of the trouble is readily understood. Where the irritation is abnormally great, as from bad teeth, the result of syphilis, rachitis, or carious teeth, it is quite clear that stomatitis ul-

cerosa will be more apt to be developed, and when developed more intense, than in a child with healthy teeth.

That stomatitis occurs endemically in certain wards of a hospital, in certain barracks, or among a certain class of soldiers, has been known since Berjeron, Taupin, and Barthez and Rilillet. By some authors the term epidemic was used instead of endemic, and the discussion naturally arose concerning the contagiousness of the affection. The older writers thought the affection was contagious, while most of the modern writers (Bohn, Henoch, Gerhardt, and others) reject this idea. Hirsch (*Handbuch d. Histor. Geograph. Path.*) comes to an opposite conclusion, which, it will be seen, is probably the correct one. The argument used by all who oppose the contagious nature of the affection is, that all attempts at inoculation of children have given negative results. In the present state of our knowledge of infectious diseases it will be granted that a conclusion based upon facts such as have been enumerated is inadequate. The experiment made, was to take some of the secretion or pus from a surface affected with stomatitis ulcerosa and inoculate the gums of another child with it. The result being negative, the disease is not contagious. We are now fully convinced of the fact that it takes more than the presence of a virus to produce a given disease. In this connection it is necessary only to refer to the experimental attempts made to inoculate typhoid-fever germs or cholera, which have so often been attended by failure, and which, when done in the correct way, are followed by success. So it is with stomatitis ulcerosa. Given a patient whose gums are in a proper condition, and inoculate these gums with pus from a stomatitis ulcerosa, and the result will be stomatitis ulcerosa. The trouble in making this experiment is that we are not in a position to state positively that in a given case the gums are in such a condition as to be affected by the virus. If we take healthy children and try to inoculate their gums with this poison, the

result will always be negative. In some researches which I made five years ago this was proven to my complete satisfaction. In making these experiments upon healthy subjects, I never succeeded in producing anything more than a slight inflammation, which got well very readily. It must be confessed, furthermore, that positive results which were obtained upon sick children were the exception and not the rule. But this was due to the difficulty of choosing proper subjects. The patients that I took, in whom I expected to get results, were affected either with rickets, so-called scrofula, or had very bad teeth with swollen gums. In all the cases in which I tried but three were successful. In these three cases there was present in one tuberculosis, and in the other two nothing more than carious teeth, with a very bad condition of the gums. The great objection which could be raised to this series of experiments is, that the patients with whom I succeeded were under the same hygienic influences as those that had the disease. In the first case, one other member of the family had the affection, and the other two belonged to the same family, and were inoculated from material taken from a third member of the same family. It may be urged that all of these three patients might have had the disease even if they had not been inoculated. However, the stomatitis followed so quickly after inoculation began at the spot where the pus was introduced, and the patients had been exposed to the bad hygienic conditions for so long a time, that the observer could not but be impressed by the fact that the disease followed the introduction of the poison into the diseased gums. I am far from accepting these results as conclusive, as I wish to extend the observation, hoping to succeed by inoculating pure cultures of the bacteria found in stomatitis ulcerosa upon proper soil. But of this much I am convinced, that it takes more than bad hygienic conditions, poor air, etc., to produce a stomatitis ulcerosa. Again, for prophylactic purposes, it is of the highest importance to remember that the

possibility exists of having the disease transmitted from one member of the family to another. It is not an uncommon observation to have more than one member of the same family affected by this disease. I have seen all the children in a single family—seven in one instance—in various stages of stomatitis ulcerosa. When we see how, for instance, for the causation of alopecia areata, Lassar (*Therapeut. Monatshefte*, ii., 1888) shows that the use of the same hair-brush, or going to the same barber, can be accepted as evidence of the infectious nature of that affection, we are certainly justified in using the frequent occurrence of stomatitis ulcerosa in different members of the same family as an argument in favor of its being infectious. For stomatitis ulcerosa we have even more direct contact than is proven for alopecia areata,—kissing, using the same table utensils, etc.

That the soldiers alone, and not the officers, become affected with this disease, when it becomes epidemic in garrisons, has been used as an argument against the non-infectious nature of stomatitis ulcerosa. It is claimed that the soldiers are under worse hygienic effects than the officers. That they come into more intimate contact with each other; that they wash out of the same basin, use the same drinking-cups, sleep together in large rooms, etc., is lost sight of altogether by those who insist upon the disease not being infectious. The officers, on the other hand, do not live together as do the soldiers, and, therefore, cannot infect each other as the soldiers do. Again, the rarity of the disease among seamen has been alluded to by Berjeron, and has been ascribed to the fact that the air upon the ocean is better than upon the land, therefore soldiers have the disease more frequently than sailors. The explanation for the comparative rarity among seamen is not the proper one, but the fact that all governments have been careful to regulate and train their sailors in such a way that they may escape that much-dreaded disease of the sea, scurvy. In doing this, espe-

cial attention is called to the condition of the mouth, and when any disease occurs there it is immediately looked to. Furthermore, all those means employed to combat scurvy—good nutrition, good air, cleanliness of the mouth—are excellent means to prevent the development of favorable soil for stomatitis ulcerosa. Unfortunately, for a conclusive decision of this matter, experiments are still wanting as to the exact nature of the poison and the nature of the soil.

The disease occurs principally between the ages of five to ten years; it is rare after this time, and very rare before the age of four to five years.

Pathological anatomy.—Bohn was the first to call attention to the fact that in this disease we are dealing with a process which Virchow calls necrobiosis. It is not necrosis, because, as Virchow states, the conception of the necrotic process implies more or less retention of the external form of the organ or tissue involved. In the necrobiotic process we have to deal with a process which usually ends in softening, and in which there is molecular or cellular necrosis, so that the tissue becomes more fluid in its consistency and more movable. (See Virchow, "Cellular Pathologie," p. 402, 1871.) If we examine the products of stomatitis under the microscope, we find very few evidences of the cells of the invaded tissues, but a molecular detritus mixed with lower forms of life, and here and there pus-cells. The process does not respect any part of the mucous membrane upon which it may be located, so that, while it begins upon the surface, the deeper structures of the gums, including the periosteum, are not infrequently invaded. When the process is most intensely developed, necrosis of bone is the result. I have in my possession the alveolar process of the lower jaw containing the four incisors which had to be taken away from a child affected with stomatitis ulcerosa. At times the process produces a complete loosening of the teeth, and when these are extracted the disease becomes tractable. At other

times the periosteum is more extensively affected, and small sequestra are separated; again, the pathological change is so extensive as to involve one whole division of the bone. In all the specimens, however, that I have been able to examine, there was no caries of the bone; as if the stomatitis had been unable to attack osseous tissue. The necrosis was evidently due to a stripping up of the periosteum, and as the alveolar process is not attached to the jaw with any great amount of firmness, being, as Hunter expresses it, "a part of the teeth," its detachment without caries could be readily explained as far as its lower border was concerned. Laterally, however, as far as the researches in embryology teach us, the detachment must have been the result of an ulcerative process, therefore caries.

The affection always begins upon the gums and in a specific locality,—at the free border. Thence it extends, as has been stated above, in all directions, causing the destruction peculiar to it. But the parts which lie in apposition to those primarily affected are apt to become infected, yet in such a manner that the process never extends beyond the buccal cavity.

Symptomatology.—Stomatitis ulcerosa begins with swelling, injection, and loosening of the mucous membrane about the teeth. At first the swelling will be observed only at the lower part of each tooth, so that the outline of the gum is altered, but not very much. Gradually the swelling increases and the mucous membrane begins to cover the lower portion of the tooth, so that the outline, instead of being curved, becomes almost straight. In the beginning the gums are affected only in so far as they form a covering for the teeth, leaving the spaces between the teeth unaltered. These spaces represent the hills of the natural curved outline of the gum, the mucous membrane covering the teeth representing the valleys. As the latter swell up, they come to a level with the elevations, producing an appearance almost pathognomonic for stomatitis

ulcerosa. The swelling may be so great as to produce a slight eversion of the part affected, and is always accompanied by injection, which gives to the mucous membrane a livid appearance. The overfilling of blood is so great that, as a rule, bleeding takes place, frequently produced by the slightest movement of the jaw, or by pressure, such as is produced by touching the gums during the act of examination by the physician. As a rule, the disease is confined to the anterior aspect of the gums, but when certain symptoms are present the careful physician will examine the posterior aspect as well. The rule certainly is, that the disease begins upon the anterior aspect; that there are exceptions is more than probable. In bad cases, however, both anterior and posterior portions of the gum become the seat of the disease. Very soon, accompanying the eversion, the gums are detached from the teeth, and sometimes before the process develops further they can be pulled away from the teeth with very little force, leaving exposed a cavity, which is filled with a peculiar muco-purulent secretion.

Even at this stage the yellowish seam at the top of the swollen outline of the gum may be perceptible. This is due to the molecular destruction which has already begun, and its presence makes diagnosis easy. The yellowish seam is at first very narrow; it may grow to a broad band, involving almost the whole of the gum.

Accompanying these symptoms the patient has a great deal of saliva pouring from his mouth. There is no disease in which salivation is so great as in stomatitis ulcerosa, and, in my experience, it is the most constant symptom. It also gives us an index to the completeness of our cure, and no case should be discharged until the moisture in the mouth is normal. Another symptom is the fetid odor of the breath and of the mouth; this arises directly from the diseased surface, not from the saliva. When the latter is collected, and great quantities can be easily obtained, it will be found, in the majority

of cases, to be odorless. Only in very bad cases, such as will be described, does the saliva also have a penetrating fetid odor. Curiously enough, this disease produces few general symptoms, and, especially in older children, little is complained of by the patient. Frequently the patient is brought to the physician on account of the fetid odor or on account of the salivation. In very young children the subjective signs are usually better pronounced. The child becomes fretful, cries a great deal, refuses to eat, has slight elevation of temperature, sleeps badly, and very soon begins to lose flesh. I have seen symptoms produced in this class of patients which would lead to the assumption of a much more serious affection. In several cases the whole disposition of the child seemed changed ; instead of a good-natured, healthy, and contented baby, there was a fretful child, crying all the time, and a look of distress and fatigue on its face which seemed to bode evil developments. One patient cried for days from pain, almost incessantly through the twenty-four hours, only dropping off to sleep from sheer fatigue. By proper treatment the whole clinical picture cleared up in a very short space of time. Parents who have once seen an attack of stomatitis ulcerosa are quick to recognize a repetition, and, having seen the good effects of remedies, are just as quick to apply them.

The lymphatic glands take part, and swell up ; they are usually soft, and remain swollen until the process has come to an end. Frequently these glands continue to be enlarged long after the disease has run its course ; rarely do they take active part so as to be inflamed, although the suppuration of the glands under the maxilla may occur.

In the various conditions described the disease is readily conquered without any active interference except the administration of remedies. When this condition is overlooked a further development usually takes place. Although a subacute or chronic form must be recognized, in which these symptoms last for an almost indefinite length of time, yet such

cases are exceptions. In the further development of the disease the essential feature is the coming to the foreground of the necrobiotic process and the production of ulcers.

If we now examine the mouth we find the yellowish, soft seam mentioned before increased in size and resting upon an ulcerated surface. When the yellow material is removed with cotton there is beneath it denuded membrane, swollen and bleeding readily, whose boundary, in its turn, is marked by injection even greater than the rest of the mucous membrane. Upon this denuded surface there is a goodly quantity of pus, but the yellowish material is very adherent to the ulcerated surface. The pus may be formed in sufficient quantity to pour down between the gum and teeth, so that when pressure is applied quite an amount may be forced up, considering the size of the affected portion. With these various changes the gum is becoming more and more detached from the teeth, so that the latter may become loosened. The process, if left to itself, continues in the same manner, the seam becoming a broad band, the ulcers going deeper, until, finally, the whole tooth is denuded. Necrosis of the bone now takes place, in either one of the ways described before. When a large portion of bone has become necrotic we look in upon a comparatively extensively ulcerated surface presenting the characteristics above mentioned. In very bad cases the possibility of necrosis must be borne in mind, and the examination is not to be considered complete until the presence or absence of dead bone has been established. Infection of other parts of the mucous membrane of the mouth also takes place after the ulcers have developed. Infection follows as the result of direct contact, and in the majority of cases affects the lower lip, then the cheeks, the tongue, and the upper lip. These ulcers are the same in every respect as those formed upon the gums; they begin with injection, then comes the formation of detritus with ulceration, the latter having the peculiar ten-

cies described before, of which the principal one is that the process does not respect the character of tissue upon which it happens to develop. In this state the lymphatic glands are still more enlarged and frequently very tender upon pressure, although rarely inclined to suppurate. Salivation has now reached its maximum and the odor is so very offensive that a child with this affection may taint the air of a whole room, or, when in a ward, it will be found necessary to use disinfectants to neutralize the extremely penetrating fetor. The ulcerative process, instead of extending by apposition, will sometimes spread directly *per continuam*, so that we may find it in the fold of membrane joining the lower lip to the lower gum. Or there may be a space of comparatively healthy tissue between the ulcer upon this fold and the ulcerated gum which, it seems to me, can only be explained by taking the infectious nature of the process into consideration. By gravity the secretions from the diseased tissue have dropped into this fold, they have remained there, and, after a sufficient length of time has elapsed, they produce the same process here that has occurred before. Reverting to what has been said in connection with etiology, it seems that the way in which this disease spreads to the rest of the buccal cavity from the gums is proof positive of the infectious character of the disease. In all cases it spreads by inoculation, however it may be accomplished; if the process is auto-inoculative it certainly is rational to suppose that, in a given patient in whom the same conditions exist as in the person affected, transmission of the affection is a possibility.

Upon the whole, stomatitis ulcerosa begins most commonly about the lower incisors; although there is no tooth about which it does not begin. In the very great majority of cases the disease first affects the teeth of the lower jaw, although this is a rule to which there are some exceptions. The teeth, in severer cases, suffer most from the disease; they are denuded,

detached from their periostem, fall out or are pulled out by the patient, who finds no difficulty in doing this on account of their being so much loosened.

Restitutio ad integrum may occur at almost any period of the disease, either as the result of treatment or, more rarely, spontaneously. When this does occur the fetor begins to disappear, the pulpons, yellowish mass is thrown off, the ulcer beneath it begins to clear up, and a new epithelial covering is formed over the place which was affected. When the bone has been affected there is more or less permanent loss of tissue; when a great portion of the alveolar process has been destroyed there remains a permanent loss of teeth, as both the temporary and the permanent teeth have been removed with the sequestrum. Rarely does it occur, as has been mentioned before, that the affection becomes chronic. It is more common for the affection to begin in a mild degree and remain for a great length of time; beginning, if such an expression might be used, as a chronic disease. These cases are characterized by a milder course, each symptom being less developed. The process does not cause the ravages that follow in the acute cases, the fetidity of the breath is not so noticeable, and is sometimes only present at certain times of the twenty-four hours, during the night or in the morning. I have never seen necrosis follow in any of these cases,—a statement which is also made by Bohn,—and, upon the whole, these cases are identical in their clinical appearance with that form of trouble which dentists call “shrinkage of the gums.” They are not so easily managed in regard to time, but constant treatment usually overcomes the affection. Relapses are the rule, but these, with ordinary watchfulness, are also readily cured.

The differential diagnosis is easy in every case. It frequently happens that aphthæ are developed at the same time with stomatitis ulcerosa, but if the clinical picture of both affections is kept in sight it is not difficult to say which spot

is aphthous and which is that of stomatitis ulcerosa. As between these affections, the decision will always be easy except in the beginning, when a small aphtha develops just upon the same place where stomatitis ulcerosa begins. This, manifestly, would be a very rare occurrence, and the difficulty could exist in the beginning only; as soon as the aphtha is well developed all doubts as to the nature of the process would disappear.

Prognosis is influenced by three factors,—the disease upon which stomatitis ulcerosa depends, the stage of the affection when the patient comes under treatment, and, lastly, the treatment itself. When stomatitis ulcerosa is caused by rickets, scurvy, or syphilis, it rarely gets well until the constitutional affections are removed. The form of rickets which predisposes especially to this affection is the so-called acute form, which, however, is supposed to be scurvy in young children (Barlow, Rehn). Here we have the worst forms and the most intractable. One fact must not be lost sight of,—viz., that stomatitis ulcerosa may become *noma* (*stomatitis gangrenosa, cancrum oris*). On account of this fact every case of stomatitis ulcerosa should be most carefully watched, although this danger exists only for debilitated, so-called cachectic children. When necrosis of bone exists the prognosis is changed from that of an inflammation of the mouth to that of bone disease. However, even here the prognosis is not very bad when the condition is recognized, because it can be readily remedied by surgical means.

The *treatment* is both prophylactic and curative. It is necessary to remove all predisposing causes when possible. This consists in improving the general condition of health in every respect,—good air, good food, cleanliness. When a case occurs in a family the other members must be protected from contagion. It is best to do this in all cases, notwithstanding the fact that the liability to affection must be very small be-

cause of the predisposing conditions necessary to produce the disease. When other members of the family are in a debilitated condition from any cause whatsoever, these precautions are especially demanded. In such cases it is well to give a mouth-wash of chlorate of potassium to the uninjected, and warn them not to use any utensil which has been used by the patient. In this way the spread of the disease is easily prevented.

There is hardly any disease which comes under our observation of which it can be so positively stated that a cure is accomplished by drugs as in the case of stomatitis ulcerosa. We have a remedy which can be looked upon almost as a specific. Chlorate of potassium given internally, and administered in this way purely for the sake of convenience, acts in a definite, well-observed way, and, with few exceptions, renders all other medication unnecessary. It is best given in a three-per-cent. aqueous solution, with a little syrup, of which from one-half to one teaspoonful may be administered every two hours, depending upon the age of the patient. There are only two objections to this remedy: one, the toxic effects which have already been mentioned, and the other the pain that is produced when it passes over the inflamed surface. I know of no means by which the latter can be prevented; cocaine has its decided disadvantages, besides overcoming the pain only partially. Fortunately, this manifestation only lasts a short time (from thirty-six to forty-eight hours), and is a positive index to the curative effect of the drug. When the chlorate of potassium produces its specific effects the symptoms usually clear up in a peculiar manner. After the remedy has been taken for from twenty-four to thirty-six hours the salivation begins to diminish materially; when the patient's mouth is opened it will still be found full of saliva, but it no longer flows out of the mouth. With the cessation of salivation the fetid odor disappears, and in a comparatively short time,

usually within a week, all the symptoms have disappeared. Now comes the time when the patient must be watched most, on account of the danger of relapses; any evidence of ulceration, be it ever so slight, demands a continuation of treatment or, frequently, the addition of some other remedy. A continuance of treatment, however, in mild or moderately severe cases forms the exception, not the rule. When ulceration does not disappear completely the cause must be found for this exceptional condition. This will usually be a carious tooth, which must be removed; if a permanent tooth, it must be treated by dental means, and if this does not stop the ulceration recourse must be had to cauterization of the gum with silver nitrate, as described in connection with the aphthous process. Where there is necrosis the sequestrum must be taken away—the sooner the better, as the process, although controlled by the chlorate of potassium, will break out afresh—or the patient's life may be jeopardized. In some cases no apparent cause exists for the keeping up of stomatitis ulcerosa; in these cases good results are obtained by the frequent use of potassium permanganate, applied with a brush.

The chronic cases do not respond to potassium chlorate as quickly as the acute ones. Even here, however, we get excellent results when combined with the local treatment just described. Nitrate of silver applied three times a week will destroy the specific process, care being taken not to touch more than the part affected, and after three or four weeks of treatment it will be found that the teeth become more firmly attached again and the patient restored. Unfortunately, relapses are even more common in this form than in the acute; these, however, will yield to the treatment just as readily as the first attack.

For the acute form the chlorate of potassium has been so completely satisfactory that other remedies, such as salicylic acid, salol, listerine, thymol, etc., will rarely become necessary,

especially if the potassium permanganate is used. It is hardly necessary to add, although highly important, that in order to prevent relapses the general condition of the patient must be looked to, although in the attack itself the administration of tonics or reconstructives seems to have little or no effect.

V.

STOMATITIS GANGRENOSA.

STOMATITIS GANGRENOSA is a disease which may occur at any time of life, but is most commonly a children's affection, being found most frequently between the ages of three and seven years. In German it is called noma, which term is employed by the French, although they also use stomatite gangreneuse. English writers speak of the affection as cancrum oris, gangrene of the mouth, gangrenous stomatitis, and noma. Up to the present time we are not in possession of sufficient accurate knowledge to say definitely whether any gangrenous process which occurs upon the gums and cheeks is to be called cancrum oris or not. From clinical evidences it is most likely that a specific process goes on in those cases which are called noma, although it would be, manifestly, improper to exclude any gangrenous process from this classification until the cause of the whole process is accurately determined. Some knowledge has been recently contributed to the etiology of this disease by Lingard (*Lancet*, 1888) and Ranke (*Jahrb. f. Kinderheilkunde*, III. xxvii., 1888), which permits us to hope that in the future this disease will be better understood than it is at the present day.

The affection is a comparatively rare one; in my own experience one case in about five thousand. Ranke (*loc. cit.*) states that in eleven years in Munich, seeing from four thousand to five thousand children yearly, he has seen only two cases (this in dispensary practice). The disease is a little more common in hospital experience, as bad cases are more frequently seen in hospital practice; in private practice the frequency depends very much upon external surroundings.

When we speak of stomatitis gangrenosa we mean a gan-

grenous process which begins upon the gums or inner side of the cheek, spreading with great rapidity so as to involve the whole substance of the cheek, and extending more or less to the surrounding tissues.

Etiology.—The first question to be answered is as to the infectious nature of the disease. There are a great many instances upon record in which the process has occurred in one ward of a hospital, in several members of a family, etc., so that evidence in this direction exists of the infectious nature of the affection. Again, on the other hand, every one who has seen any cases at all will have seen such in which it was impossible for the patient to have come into contact with any one who had had a gangrenous process of any kind. The experience of any one individual is too small to decide this question definitely, yet I venture to state that most physicians would answer it negatively. There are many things which speak in favor of the infectious nature of the disease: that it has occurred as stated above; that the process usually occurs, primarily, at the junction of the skin with mucous membranes, the anus, the genitals, the mouth, where infection takes place most readily (Gerhard); that the disease is found in affections in which the mucous membranes are in the best possible condition for the reception of infectious material. But proof, except by inference, is still wanting. There can be no doubt of the fact that *cancrum oris* occurs spontaneously without the existence of previous cases.

No disease exists in which the predisposing cause is accepted so universally as in *stomatitis gangrenosa*. This predisposition consists in a reduction of health in a way which, for the present, cannot be accurately defined. Ranke says concerning it, "We know that other carriers of infection require a certain amount of predisposition in order to develop their functions. In this direction the supposed virus of *noma* would have to be placed at the extreme end of the list of

infectious agents. It would be necessary to premise that its attacks upon a healthy cell would be absolutely futile, and that it can manifest its effects only upon extremely-weakened cells." From a clinical stand-point we know that the weakening of the cells is produced by certain diseases : the acute exanthemata, especially measles (forty per cent. of all cases, Barthez and Rilliet), long-continued fevers and infectious diseases, typhus, whooping-cough, syphilis and scorbatus, chronic intestinal catarrhs and malaria. To this must be added the excessive use of mercurials, although this causative factor was very much overestimated in former days. Bohn has shown the importance of stomatitis ulcerosa as an etiological factor for noma, possibly as its forerunner, and therefore of great importance ; but the connection between the two diseases in all probability ceases there. Malaria is looked upon by Hirsch as of prime importance, simply from geographical evidence ; and while it cannot be denied that the reduction of general health may be produced by malaria, the direct cause must be found in something else. In general terms it may be stated that all those predisposing causes which have been enumerated under stomatitis ulcerosa are accepted for stomatitis gangrenosa. The fact, however, must be insisted upon that predisposition is so important that in any given case of noma the general condition of the patient would immediately be noticed by any physician, and would be a source of alarm upon very cursory inspection.

Investigations as to the direct cause are as yet in their infancy. Cornil and Babes make the statement that short streptococci are found in stomatitis gangrenosa, and, in certain cases, rods like those of pulmonary gangrene. No allusion is made to cultures or inoculations by these authors. Ranke (*loc. cit.*) has found streptococci resembling those described by Koch as producing progressive tissue-necrosis in field-mice. He has made no cultures, but has inoculated rabbits with

pieces of tissue taken from the immediate neighborhood of the gangrenous process. The animals died, but in no instance was he able to produce gangrene, so that in his conclusions, at the end of his paper, Ranke states that "up to the present the specific nature of the cocci present in canerum oris has not been proven." Lingard (*loc. cit.*) found bacilli in thread-like growth 0.004 mm.-0.008 mm. long, 0.001 thick. Cultures were made as well as inoculations. Young pigs and calves were killed by these inoculations on the tenth and eleventh days, and septic lesions of the heart were produced. No statement is made concerning the production of gangrene except that the lower forms of life were also found in certain petechial spots in the human subject. It is difficult to judge of these results, as the heading of the article is "Cancerum Oris or Ulcerative Stomatitis," terms which, according to our view, are not synonymous. As, however, the term cancerum oris is so distinctive it has seemed to us that only the gangrenous process could be referred to.

It would be rash to try to bring these various observations into accord, the one with the other; as a *résumé* it might be well to state that all these observers have found lower forms of life in noma. Ranke has found them within the tissues; Lingard has cultivated them; both have killed animals, the one by inoculating them with the tissue, the other by injecting cultures. In no instance was there produced anything resembling the pathological changes of noma, although Ranke introduced a piece of diseased tissue under the mucous membrane of the mouth. It is impossible to state whether the poison has been isolated by Lingard or whether he has found the virus of something else. His description does not correspond with the one given by Frühwald (*Jahrb. f. Kinderheilkunde*, II. xxix., 1889) for a bacillus found in ulcerative stomatitis, and does not agree with the pictures seen by Ranke. Knowing, as we do, the absolute importance of a predisposing cause, it is futile to

discuss the method by which the direct cause acts before this cause has been isolated, so that the question cannot be definitely answered whether it comes from within the system as a poison of some sort, or from without as a lower form of life.

In the very great majority of cases some lesion is found upon the mucous membrane of the mouth which precedes the attack of gangrene. In some instances no lesion could be found, but on account of the locality of the process it has been found impossible to exclude such with absolute certainty. The case which has often been quoted from Gierke's article (*Jahrb. f. Kinderheilkunde*, N. F. 5, p. 269) as opposed to this view cannot be considered in this light, as the gangrenous process evidently arose from a stomatitis ulcerosa. The appearance of gangrenous spots upon the skin, in this case, could be readily explained if the assumption of a specific virus is accepted.

Pathological anatomy.—The process is one of rapid phlegmonous gangrene. Around that portion which has been destroyed there is found an infiltrated zone (Ranke, *loc. cit.*). This is characterized by true necrobiosis; all evidence of pre-existing tissue has disappeared under the microscope; in its stead there is found a perfectly homogeneous substance which shades off in the direction of the adjacent tissue. This homogeneous substance is already dead, and around it we find the connective tissue increased, its corpuscles in cell division and its blood-vessels closed by thrombi. The micrococci are found both in the homogeneous as well as in the proliferated tissues. Ranke has made interesting observations concerning the karyokinetic figures which, as he states, are found both in the fixed as well as in the wandering connective-tissue corpuscles and in the muscle-cells within the proliferated zone.

Symptomatology. I. *General.*—These symptoms vary very much, depending upon the disease upon which noma is ingrafted, for a healthy child cannot be attacked by noma. It may be stated that the intensity of the general symptoms is

in direct ratio to the severity of the disease. A great many cases are upon record in which the children seemed in the beginning to be very little affected by the development of noma. Bohn gives a description of this condition which leads one to infer that the children, in this disease, are rather cheerful than otherwise. While the fact exists that frequently patients will be attacked by stomatitis gangrenosa and pay very little attention to the local process, pulling out loose teeth, picking off pieces of gangrenous tissue, etc., in a very short time general symptoms supervene which show that we are dealing with a process which produces a very deep impression upon the general condition. Fever may not be present in the beginning, but develops sooner or later, reaching 104° to 106°, becoming hectic, especially when suppuration is present, and before death the temperature frequently falls to subnormal. The pulse usually follows the temperature, but throughout is weak, easily compressible, and small. Diarrhoea is present in almost every case. This diarrhoea is of the most intractable variety, and, as Gierke has pointed out, must be due to the swallowing of material from the diseased surface in the mouth. Lesions in the organs are also common, especially catarrhal pneumonia, probably due to the entrance of septic material into the bronchial tubes. Diphtheria has been observed in several cases (West, Gierke). As a result of the general infection, the local symptoms, the fever, the diarrhoea, death usually comes to the patient by exhaustion. The children then become apathetic, refuse all nourishment, are restless, and finally die in collapse. The nervous system is rarely implicated even in the worst cases.

II. *Local.*—The local process usually begins suddenly : if the result of a stomatitis ulcerosa, the symptoms of ulceration are changed to those of gangrene ; if upon a comparatively healthy mucous membrane, the physician can never be in doubt as to the nature of the process. It is essentially a moist gan-

grenous process and characterized by all the symptoms of this condition. The beginning of the process is to be found, usually, upon the gums or upon the inner surface of the cheek, near the corner of the mouth, and, it is said, more frequently upon the left than upon the right side. Possibly the first thing that will strike the observer is the appearance of the peculiar odor of gangrene; if stomatitis ulcerosa has preceded the development of noma, the fetor of the former disease is covered over by the intense and penetrating odor of noma. Upon examination there will be found at the point of development an ulcer, gangrenous, which spreads with great rapidity. Very soon the cheek begins to swell so that if taken between the thumb and forefinger it will be felt to be thickened throughout its entire structure. This swelling is more or less oedematous, the skin becomes waxy, and in a very short time, sometimes within twenty-four hours, the whole side of the face up to the eyelids and down to the jaw or upon the neck becomes involved. This cheek may be painful upon pressure, but more commonly the patients do not complain of painful sensations. If we now look at the ulcer within the mouth we see that it has grown very much in depth, evidently eating its way through the substance of the cheek. As it comes near the integumentary surface, symptoms of its approach begin to manifest themselves upon the skin. The latter becomes discolored, red, blue, purple, black, or a combination of several shades. The reddish tint is usually observed in the beginning, and the spot of gangrene may be surrounded by a red areola. When the gangrenous process is completed there is always developed a dark spot. In a great many cases a bulla is formed, over the spot to become affected with gangrene, filled with ichorous fluid. The epithelial covering breaks and, with this, perforation of the cheek takes place. In case the bulla has not formed, melting away of the tissues takes place in one direction only, from within outward, the skin then may

become mummified, but is finally softened and breaks down. Rarely is the gangrenous process completed when perforation has taken place ; in one case which came under my observation, resulting from chronic malaria, there was what appeared as a cleanly-cut, oval hole. The rule is that the process now extends, involving the soft parts of the cheek, going down upon the neck, eating into the nose, the eyelids, affecting the frontal bone, destroying the eye, but rarely extending to the other side. In the mouth the devastation is apparently greater than upon the surface. While we find the destruction within the mouth to be very great in all cases, upon the surface it may be comparatively limited. Nothing is spared ; the bones are denuded, the teeth loosened, the tongue and hard palate may become affected, even the soft palate and the tonsils may become involved. The whole is converted into a black, fetid, pulposus mass. The patient may now be considered in a frightful condition, and there is hardly any sight so repulsive as a child with well-developed nomia. If to this appearance there is added, as is not infrequently the case, the entire apathy of the child for the local condition, we have a combination which calls for the utmost sympathy on the part of the surroundings.

With all these changes the patient complains little of the local condition. The flow of saliva is very much increased ; at first the patient swallows very well, but ceases to do this as the disease progresses. Again, the appetite may not be diminished ; but this also disappears in a short time. The odor that fills the room is frightful ; the whole house is sometimes filled with it, so that the diagnosis of gangrene can be made as one enters at the front door. Hemorrhages are quite rare on account of the fact that all the blood-vessels are closed by the thrombi.

The course of the disease tends either to death or, what is very much rarer, recovery, either spontaneously or as the result of treatment. When death comes it is as the result of the general condition. When spontaneous recovery takes place

we find a line of demarcation around the gangrenous spot, the surface is finally converted into one covered by granulative tissue, and there takes place cicatrization, leaving frightful scars. This is also very rare. In most cases that have recovered it seems that the treatment has had something to do with the result. Relapses take place, but they are comparatively rare,—two cases out of twenty. (Gierke.)

The duration of noma varies from one to two weeks, but sometimes very much longer. Perforation of the cheek has taken place in as short a time as twenty-four hours, but usually takes three or four days.

Prognosis.—It is almost useless to discuss the factors which go to make up our prognosis in a case of noma, as nearly all cases die. The mortality is given as ranging from seventy per cent. to ninety per cent. of all cases affected. The statement can be made that the more intense the local process the greater the mortality. This seems paradoxical, yet the fact must not be lost sight of that when gangrene ceases, the patient still being alive and not affected by complications, the general condition upon which the final result depends must certainly become improved. Complications, especially catarrhal pneumonia or diphtheria, will render our prognosis absolutely unfavorable.

Treatment. I. Prophylactic.—Unfortunately, little can be done in this direction. The disease may develop when and where it is least expected. Its development is very sudden, and, as has been stated, it may develop in patients whose mouths are apparently perfectly healthy. On account of the rarity of the affection the physician does not think of noma, and, fortunately, this is not necessary. In hospital practice the careful watching of individual cases, their possible isolation combined with antisepsis, are certainly of value. The modern hospital, however, can hardly be charged with epidemics of noma, at least there are none such upon record.

The treatment of a case which has developed can resolve

itself into two principal divisions,—1, the general; 2, the local treatment.

Of the general treatment little need be said. The disease is found in reduced subjects, usually in such which have been worked at by physicians precisely in that direction which seems needful for the cure of noma,—the improvement of general health. The indications in every case are to keep up the strength of the patient until it has become possible to make the attempt to cure the local process. The tonics and stimulants would come into play here, but always with the needed precaution not to disturb the digestion. The patient must be fed with condensed, nutritious food, if necessary predigested.

The local treatment has resolved itself to an artificial limitation of the gangrenous process by substituting an artificial destruction of tissue. For this purpose a great many substances have been employed. It is essential that the remedies be used as early as possible. Barthez and Rilliet, as well as others, state that the caustic ought to be used “before the deep tissues of the cheek are invaded.” The caustics which seem to enjoy the greatest reputation are hydrochloric acid, then nitric acid (West). Evanson and Maunsell report good results from the local application of sulphate of copper in six-per-cent. solution. The same authors also speak highly of sulphate of zinc in twelve-per-cent. solution. But it has always seemed to me that if anything is to be accomplished by treatment at all, in this disease, we ought to have recourse to those remedies which act quickly, deeply, and thoroughly. For this purpose caustics must be used whose action is intense, destroying that with which they come into contact and producing distinct reaction. These caustics can be divided into chemical and thermal. Of the latter class we have the white-hot iron, the galvano-caustic wire, and the Pacquelin cautery. The chemical caustics that have been used are either in solid or fluid form, and nearly every chemical has been used that has caustic prop-

erties. Of the thermal caustics it has been said that their application is difficult and their action inexact because we could not tell where to find healthy tissue. The same objections (Bohn) have been raised against fluid chemicals, and Bohn therefore recommends nitrate of silver in stick. The great advantage of nitrate of silver, applied in this way, is that it does not attack healthy tissue more than seems necessary, but destroys all that is dead or becoming gangrenous (just as it acts in *lupus vulgaris*). The only objection to the use of nitrate of silver is whether its action upon the healthy tissue is sufficiently energetic to produce any benefit. If the indication is to cause deep destruction of healthy tissue, so as to produce demarcation well marked, the nitrate of silver cannot be relied upon. It is certainly a fact that some cases of noma will get well spontaneously : the only case I have ever seen recover did so with applications of a solution of permanganate of potassium without the use of any cautery, and therefore not all cases of recovery are to be attributed to the remedy used. In the two cases of recovery of Gierke, chloride of zinc and pyroligneous acid were used ; Foerster (*Jahrb. f. Kinderheilkunde*, v. p. 328) reports a case which was cauterized by using dilute muriatic acid, then nitrate of silver in stick, and finally in solution. The results of Evanson and Maunsell, referred to in high terms and corroborated by J. Lewis Smith, certainly show that some patients will get well without very much treatment, certainly without cauterization. As far as I am concerned, I would not like to take the risk of treating any case of noma without the use of a caustic of some sort. It seems to me that with the very bad prognosis, *quoad vitam*, the destruction of a little more or less tissue ought not to be taken into consideration at all, especially when we bear in mind that much more tissue is already dead than appears when we judge by the classical gangrene color only. The object of cauterization must be to destroy not only the waxy zone, but to

go into the tissue, for some distance, that seems perfectly healthy, if we can dare to hope for setting up a process of repair, or closing up the lymphatics against further invasion.

If circumstances permit, the patient should be anæsthetized before the caustic is employed. This is frequently inexpedient on account of the great weakness of the patient. Before the caustic is applied all the necrotic tissue should be removed with forceps and scissors, and then the operator is ready. The galvano-caustic wire or Pacquelin's thermo-cautery seem to me to offer advantages that are not afforded by any other means. Their action can be limited, they can be made to act as deeply or as superficially as the operator may choose. It is necessary to take into consideration that when the cautery is applied when it should be, the inner surface of the cheek is the place where it will be used most frequently. It will immediately be seen how readily this can be done with either of these instruments when it would be very awkward with a fluid acid and very difficult with any of the fluid pastes. The question of how often the tissue should be cauterized has been answered : not more frequently than once in twenty-four hours. There certainly cannot be any law put down, as every case is a law unto itself. With cauterization the ordinary antiseptic methods of treating wounds will be sufficient local treatment for most cases of noma. Sometimes poultices will be required, sometimes the granulating surfaces will need to be treated. The surgical treatment of the cicatrices ought to be put off as long as possible, as it has been found that plastic operations do not succeed very well when performed early upon patients that have had noma, and also that noma may recur as the result of these operations.

It is unnecessary to add that the outlook, with all methods, with everything in apparently good condition, remains unfavorable to the patient, do what we will and do it as we will.

VI.

STOMATITIS CROUPOSA—STOMATITIS DIPHHERITICA.

IT is not the province of these articles to enter into the discussion concerning the identity of croup and diphtheria. The opinion which is held by any individual upon this question depends upon a variety of circumstances, not the least important of which seems to be the kinds of cases which have come under his observation. Some clinicians (and their number is not small) have not been satisfied with the decisions that have been handed down by those who were formerly looked upon as authoritative,—the pathologists,—and with more or less powerful arguments, always clinical in their nature, have reasoned themselves into the view of the identity of the croupous and diphtheritic process. Others, on the other hand (and again their number is not small), who have seen what they considered as pure and uncomplicated croup cases, for which they, in their turn, have advanced clinical arguments, have insisted upon a separation of the two processes as non-identical. As we no longer look upon the pathological anatomist as the supreme judge to whom the last appeal is made, and as we certainly have no right to decide questions of etiology by purely clinical observation, the whole discussion has resolved itself into a matter of scientific belief, which, although a contradiction in terms, is of wide-spread existence. From this stand-point it may not be improper to state that the author believes that the diphtheritic and croupous processes, while frequently combined, are in their nature two essentially different entities, caused by different agents and followed by different results in the human being.

At the present day the court of appeal is made up of bacteriologists with chemists (the latter in the minority), who

have not, as yet, passed their verdict upon the mooted points. It is by no means positive that when this will occur it will be final for all time, but for us the question will be settled when this is satisfactorily done, if such is possible, by these jurors.

Stomatitis crouposa is always a complication of angina crouposa. As far as the literature and my own experience goes, I know of no case of primary croupous inflammation of the mouth. In a great number of cases of angina crouposa the membrane develops simultaneously upon the tonsils and adjacent parts of the mouth. In severe cases extension takes place to the tongue, the lips, and the cheeks (Steiner). One of the characteristics of this process is the comparatively slight involvement of the lymphatic glands. We find this in stomatitis crouposa as well as in croup in other locations. As the disease is part of another disease of very much greater importance, and as it does very little, if any, damage by itself, its prognosis can be of importance only in that it indicates by its presence the intensity of the condition which affects the patient. There is little, if any, treatment required for the stomatitis beyond the one used for the angina. When the membrane has been shed, it will sometimes be found necessary to apply some of the remedies which have already been recommended in those cases in which there exist superficial ulcers of the mucous membrane of the mouth, although these are exceptional.

STOMATITIS DIPHHERITICA.

In birds and in the calf stomatitis diphtheritica is the most common form of deposit of diphtheritic membrane. In the human being primary diphtheria of the mouth is extremely rare; yet cases do occur, and one especially is of great interest (found in Gustin, "Étude Clinique sur l'Inoculabilité de la Diphthérie," Paris, 1883, and Sanné, "Dictionnaire Encyclopédique," iii. p. 29). The case was origi-

nally reported in the *Union Médicale*, 1859, by Professor G. Séé. A wet-nurse was nursing her own child, ten months old, and a little girl who had diphtheria of the vulva and of the lips. Several days after the latter was taken sick the nurse's own child began to complain of the same series of symptoms,—*i.e.*, diphtheria of the lips,—which was followed by angina diphtheritica and croup, the latter fatal. The mother, who persisted in kissing her child, was also taken with diphtheria of the lips, but the disease remained localized to this place. Another child was also affected with diphtheria, but there was produced, from the start, an angina diphtheritica. The nipples and breasts of the mother remained normal throughout the whole course of the disease. The case is of interest not only because of the number of interesting cases connected with it, but also on account of the fact that we have three cases of primary diphtheria of the mouth. Although cases of this sort are extremely rare, yet they must be more frequent than would be inferred from the very few cases upon record. The statements made by the best authors—Jacobi, Seitz, Baginsky, and others—lead us to the conclusion that they have seen cases of primary diphtheria of the mouth, although, as Jacobi says, “diphtheria of the mouth (primary) is not very common, but is not infrequent with diphtheria of the pharynx or nose.” Sanné says that in epidemics it is not rare that the mouth is affected primarily. When infection has taken place by the mouth the lips are usually the first to become affected. From here the membrane may extend to any part of the mouth or to the tonsils.

In diphtheria of the tonsils, when the membrane extends to the mouth, we usually see the following method of invasion: first, the pillars of the fauces, perhaps more commonly simultaneous with the angina; then the tongue, cheeks, lips, and gums. This, however, is subject to a great many exceptions, for example, the case reported by Seitz (“Diphtherie u. Croup,”

1877, p. 312). The membrane was first noticed upon the left tonsil; then upon the gum at the last right incisor tooth; then upon the right side upon the dorsum of the tongue, at the same time diphtheria of the nose. Those cases are apt to be accompanied by diphtheria of the mouth in which the general infection is very great, in so-called septic cases. It is pretty well established that diphtheria cannot be inoculated upon a healthy mucous membrane (Rajewsky, Loeffler, and others), and therefore it is necessary to conceive of some alteration of the mucous membrane of the mouth before it can become diphtheritic. This is not difficult in diphtheria, and the clinical evidence will confirm all that is necessary to make a mouth diphtheritic. In all cases of diphtheria more or less profound changes go on in the mucous membrane of the mouth,—from a simple injection or dryness to a stomatitis of one kind or another. It is a matter of astonishment that stomatitis diphtheritica does not occur more frequently than is the case, when we take into consideration that the membrane which is expectorated almost always comes into contact with a mucous membrane ready to have the seed for further growth implanted upon it. The frequency of invasion of the mouth can be approximately stated by referring to a table published by Minnich ("Croup u. seine Stellung zur Diphtheritis," Wien, 1888), in which three cases of thirty-seven of diphtheria had stomatitis diphtheritica. When, however, the mucous membrane of the mouth becomes diphtheritic we always find accompanying the process a stomatitis catarrhalis, which is independent of any condition there may have been present before the diphtheria had developed.

Salivation is an almost constant symptom, accompanied by a fetid odor from the mouth, the same which is noticed in angina diphtheritica, in well-developed cases. Before the eruption of the membrane the place upon which it develops becomes very much injected, almost livid, and in a comparatively short time (from twelve to twenty-four hours) we see

upon or within this injected area the characteristic membrane. This membrane, depending somewhat upon the intensity of the process, may appear as discrete spots which afterwards confluence, or the whole livid area may immediately be covered in its full extent by one membrane. At the same time there is marked swelling of the lymphatic glands under the jaw, accompanied by more or less tenderness upon pressure. In septic cases, frequently the general condition of the patient is such that subjective signs are of no especial value, or they do not exist. The membrane remains for a variable time,—three to five or six days,—and then either drops off or ulcerates away. In either instance there is left a denuded spot, where the epithelium is absent, and, depending upon the amount of ulceration, more or less loss of substance. As a rule, cicatrices do not occur, but sometimes the loss of substance becomes great and then connective tissue is formed; especially is this the case upon the tongue. During the whole process there is a continuous flow of saliva, which may erode the skin with which it comes into contact. At the corners of the mouth it is not uncommon to find a diphtheritic patch, and sometimes the diphtheritic process will extend to the skin upon either the upper or lower lip.

Hemorrhages occur with this form of stomatitis, sometimes severe, at other times the loss of blood is not very great; they are always of bad prognostic omen, for they mean general infection of great intensity. The oozing out of a few drops of blood from the mucous membrane in a diphtheritic patient, especially when due to mechanical irritation, of course means nothing; but there are cases upon record in which the patient has lost his life directly by hemorrhage from the mouth, and a great many more in which a slight hemorrhage seemed to be sufficient to destroy the last spark of vitality left to the debilitated subject. (See Sanné, *loc. cit.*)

The prognosis is best expressed with Jacobi (*loc. cit.*), that

"under all circumstances stomatitis diphtheritica is of a dangerous nature." In primary cases the prognosis seems better than in those in which the membrane has extended from other parts. Such cases are so very rare, however, that they need hardly to be taken into consideration when generalizing upon the subject. It seems necessary to state that the localization of the membrane has very little, if any, effect upon the production of sequelæ or upon the possible development of complications.

The treatment is the same as that for any other form of diphtheria; but Baginsky's statement that this process "challenges local therapy" is one that ought to be borne in mind. Every physician who has dealt largely with diphtheria has selected for himself the local remedy upon which he places the most reliance. These are the cases in which he can test this remedy and see how valuable it is. There is no difficulty in removing the membrane, accessible as it is to all local medication, and for this purpose a great number of remedies can be used: carbolic acid, corrosive sublimate, nitrate of silver, the persalts of iron, permanganate of potassium, trypsin, papayotin, etc. The great question, after all, is whether the removal of the membrane will do the patient any good. When we are dealing with a case in which the constitutional effect is not very great, or even moderately severe, I have no hesitation in saying that the indication exists for the removal of the membrane. When, on the other hand, severe constitutional symptoms exist, the local treatment of diphtheria is futile and, sometimes, harmful. In such cases time cannot be wasted nor the strength of the patient be dissipated by attempts at destruction of the membrane. Unfortunately, whatever is done is for naught, in the great majority of such patients; but, at all events, attempts should be made to treat the general condition which is the one producing the most serious and, most frequently, the only issue,—death.

VII.

STOMATITIS SYPHILITICA.

IT is almost unnecessary to state that, strictly speaking, there is no such process as stomatitis syphilitica. Syphilis, *per se*, does not produce stomatitis except in an indirect way, in that it may either cause the mucous membrane of the mouth to become more sensitive to stomatitis producers, or in that it causes a lesion of some sort which, in its turn, provokes inflammation. The term has been retained in our classification for the sake of convenience and because it is found extensively employed in the literature of stomatitis.

Syphilis manifests itself in and about the mouth in well-defined forms and in well-recognized localities. The localities that are especially apt to be affected are the lips, the tongue, the tonsils, and the mucous membrane covering the cheeks. The teeth, it is claimed, are subject to the general law of the characteristic nature of the syphilitic lesion, but this is still open to discussion. It is quite difficult to lay down an absolute law which will hold good in every case, so that we may be guided by it for diagnostic purposes. If we hold fast to specificity of lesions produced by syphilis, there are cases in which it becomes absolutely impossible to make a diagnosis of syphilis simply by an examination of the patient. Again, if we rely upon the statements made by parents, we are in danger of erring in two directions,—either in considering a manifestation as syphilitic when it is not, or, more commonly, of overlooking a specific manifestation entirely. Fortunately, these combinations can only arise exceptionally, for, as a rule, the patient may be observed for some time, and we have collateral evidence which guides us much more safely than the admission or denial of the parents. If we except the teeth, the lesions

in the mouth are nearly all characteristic, and no doubt can arise as to the general diagnosis in their presence.

Syphilis manifests itself upon the lips in one or other of the following forms: syphilitic fissures, papules, plaques, and erosions. The fissures (rhagades) represent the most common specific manifestation upon the lips. When they are present they are absolutely characteristic and leave no doubt as to the diagnosis. They are characterized by their location, their appearance, and their duration. The most common place of appearance is the corner of the mouth, then the upper lip and, comparatively rarely, the lower lip. Upon the upper lip we usually find them upon either side of the median line, and they differ somewhat from those found at the corner of the mouth. In the latter place, as a rule, the most striking thing about the fissure is that we are dealing with infiltration which has been split in or about its middle. The fissure sometimes loses itself in the mucous membrane, sometimes stops before reaching it, sometimes runs into the mucous membrane, as in the case of the fissures upon the lips. The infiltration (small-celled) is somewhat elevated, the fissure may or may not be covered by a crust, and, contrary to most syphilitic eruptions, produces more or less pain when the mouth is opened. On account of the crack's being connected with the mucous membrane, small hemorrhages may occur, and the crust may therefore be made up of coagulated blood which has extraneous matter mixed with it. These rhagades do not secrete very much unless they are made up of papules, when their surfaces as well as the fissures are apt to be moist. This form is rarely found upon the upper lip, where the fissures are characterized by the lack of infiltration, but the fissure usually ends in an infiltration upon the mucous membrane. If we turn up a lip upon which there is such a fissure the rhagade will be found to end in some form of syphilitic lesion upon the inner surface. This class of fissures is sometimes present in great numbers, dis-

figuring the mouth and causing great annoyance to the patient. All rhagades are characterized by their persistency and by their lack of tendency to spontaneous healing. Those found upon the lip, although they do not secrete more than the form at the commissure, are even more persistent. They may cause disfigurement of the lip because of the inflammation which is caused by them, which, when they heal, always produces cicatrices. Again, the fissures may be so deep and so numerous that by their presence alone the whole shape of the mouth is changed.

Papules are most commonly found at the commissure, although the free border of the lip is sometimes infiltrated and thickened by a broad papular eruption. As stated above, they may have a fissure upon them, but usually they are found in the form of condylomata lata. Their surface is elevated and moist; their tendency is to break down in the centre, producing an ulcer which is covered by a crust. When this crust is removed there is found the ulcerated surface, which does not bleed very much. The papules, in and of themselves, produce very little pain unless they involve the mucous membrane.

Plaques muqueuses and erosions are found upon the mucous membrane. They are both superficial, but cover more space than any of the forms described before. The infiltration is not so well marked, but nevertheless there is more or less thickening present.

Upon the tongue there are found various lesions, some well known and thoroughly accepted as characteristic, others sufficiently characteristic as lesions but not as syphilitic lesions. Among those which are accepted by all are the various manifestations with which we have become acquainted in discussing the alterations upon the lips. The most common varieties found upon the tongue are: the plaques muqueuses and syphilitic ulcers. Both have infiltrated edges, but the plaques, in this situation, are raised above the level of the tongue, while

the ulcers are considerably depressed. Either one or the other form is characteristic and sufficient for a diagnosis of syphilis. Their localization depends somewhat upon the presence of an irritation, so that we find them opposite sharp teeth, but they may occur upon any part of the tongue without any especial cause being determinable.

There is no reason why the so-called primary sore could not develop upon the tongue of the child, and, certainly, such has occurred. As it would not differ from that in the adult, there is no especial interest attached to it. Children contracting syphilis from their nurses have the first manifestation either upon the lips or upon the tongue. When an ulcer which occupies a greater part or the whole of the tip of the tongue is present it is always suspicious, and when, in addition to the locality, a decided infiltration is present, the diagnosis is almost complete.

Among the manifestations upon the tongue which are frequently overlooked must be mentioned certain changes which go on in the epithelium. They are not characteristic of syphilis, but they are usually present in the early period. They consist in a loss of epithelium which especially affects the mucous membrane covering the filiform papillæ and the inter-papillary coating. As a result of this loss, the tongue takes upon itself a "shaven" appearance, it is redder and dryer than usual, and careful inspection reveals an absence of filiform papillæ with a corresponding prominence of the fungiform papillæ. The latter is only comparative, as these papillæ also have been deprived of their epithelium, but they seem to be more prominent because they are all that is left of the elevated formations of the tongue. When it is possible to get a view of the circumvallate papillæ they will be found to be very prominent and decidedly enlarged. In infants it is difficult to see the base of the tongue, but in older children it is very much easier than in adults, and in all cases of syphilis I

have found these papillæ very much enlarged, so that in some instances my attention has been especially directed to them.

There exists a condition of the tongue which has been frequently described, but which has received especial attention comparatively recently, has been named, and been made characteristic for syphilis. This condition, called by Parrot (in "Syphilis Héréditaire et le Rachitis," 1886) desquamative syphilitide of the tongue, is of great interest in all directions. Before going on to discuss its relation to syphilis and the evidences of such relation, it will be necessary to give a description of the condition. It is essentially a children's affection in the sense that it begins early in life: further observations will have to be recorded before we can decide whether it extends into adult life. I am now watching two cases in which the affection began twelve and thirteen years ago, and in neither case has the condition disappeared. I have also under my observation a young lady, now twenty-four years of age, in whom the process was first noticed when she was about five years old. The condition is not very common (fifteen cases in two thousand one hundred and ninety-seven sick children.—Parrot), but sufficiently so to enable every physician to see cases. A great many names have been employed to designate the affection: wandering rash (Berker), ringworm, the lichenoid condition (Gubler), geographical tongue, and some have confounded this form of trouble with another psoriasis, tylosis, lichen or ichthyosis of the tongue. The latter are, however, affections to be distinctly separated from that which has been brought into prominence by Parrot.

The location of the disease is "almost invariably" the dorsum of the tongue, somewhere in front of the circumvallate papillæ (Butlin, "Diseases of the Tongue," p. 161). It begins at the edge or tip of the tongue in the form of a small patch which is characterized by a more opaque and whiter color than the rest of the mucous membrane. This patch is

distinctly bounded by a greater or smaller part of the arc of a circle. The next step in the development consists in a simultaneous enlargement of the outline and a shedding of the epithelium within. The outline grows, in that the thickening of the epithelium extends, the circular line becoming greater, although usually developing upon the same radii which existed for the original patch. This outline depends upon the same pathological process as the first white, opaque spot,—viz., a too rapid formation of the epithelial cells which have not quite gone through the changes to become adult epithelium. As a result of this, these cells are heaped upon each other in greater number than normal, and, as a final result, they are more rapidly shed. The spot now assumes the appearance of a white or yellowish-white boundary surrounding a red surface. The red surface is characterized by a glossy appearance due to the covering of the mucous membrane by young and translucent epithelium and an entire loss of filiform papillæ. By extension of the boundary and the simultaneous shedding of the epithelial layer the whole or a greater part of the tongue may become involved in one patch. According to Parrot the whole process is accomplished in from five to six days; this, however, does not agree with my own observation, according to which the period of time varies so greatly that I would hesitate very much to ascribe any exact length of time for the development of a spot with cessation of growth. According to Mr. Butlin (*loc. cit.*), "as the circles widen out, so may they contract again," from which we may infer that the epithelium grows from the periphery towards the centre of the denuded spot. In the same sentence we find "but the rapidity of the subsidence is often so great that the surface of the tongue does not instantly regain its normal aspect."* The epi-

* "As the circles widen out, so may they contract again, until each and every circle may disappear from the surface of the tongue; but the

thelium, according to my observation, forms all over the denuded spot, immediately the process of extension comes to an end, and the rest of the process, so well described by the author quoted, is due to the fact that the epithelial layer is formed as such, and no longer as young cells never destined to adult development.

Frequently two or more patches develop at the same time at different parts of the tongue. When this is the case one of them ceases to grow, while the other one seems to grow more rapidly, at least in the direction where they are contiguous. Again, it will occur that one or more new patches develop within an old outline; in such cases we see an undulatory, wavy appearance of outlines and slight elevations within the first boundary line. The course of the affection is that of a chronic disease. It may disappear for a time, so it is stated by all authors, but relapses are common. In the cases which have come under my observation the tongue is never perfectly well, but there is always more or less evidence of this process, either in the form of an abnormal redness, the beginning of the development of a patch, or the presence of well-marked patches. I have taken occasion to watch the patients under my charge at times when they were otherwise perfectly well, but have always been able to see some change which would stamp the tongue as not perfectly natural. This disease either runs its own course and, after a greater or less time, leaves a normal tongue, or, as in the instances mentioned in the beginning, it lasts into adult life. How long into adult life the process may reach I am, at present, unable to state.

The pathological anatomy of the disease (Parrot) consists

rapidity of the subsidence is often so great that the surface of the tongue does not instantly regain its normal aspect; it is slightly redder and smoother than natural."

simply in an irritative process or subacute inflammation, which goes on in the derma accompanied by the production of an abnormal epithelium, both quantitatively and qualitatively. Beyond this nothing distinctive can be obtained for this inflammation from Parrot's description, and he seems to have been the only one who has made microscopical examinations of tongues affected by this disease. The cause or causes of this affection are unknown to us. Parrot's view is the one which must occupy us here, as his reason for the syphilitic nature of the "wandering rash" is as follows: "J'ai pris au hazard dans mes notes 31 cas d'affection desquamative, et dans 28 les enfants portaient des marques incontestables de la syphilis héréditaire. Cela me suffit pour la caractériser, et je dis qu'elle constitue elle-même une manifestation syphilitique, et que la qualification de syphilide desquamative de la langue, que je propose, est suffisamment justifié" (p. 132, *loc. cit.*). ("Hazard, I have taken 31 cases of the desquamative affection from my notes, and in 28 the children showed incontestable signs of hereditary syphilis. This is sufficient to characterize it, and I say that it of itself constitutes a syphilitic manifestation, and the name which I propose, desquamative syphilide of the tongue, is justified sufficiently.") Again, on the next page he states that from the 1st of January to the 30th of April, 1881, two thousand one hundred and ninety seven children had entered the hospital. Of these, three hundred and twenty-eight had hereditary syphilis; out of the whole number of children, fifteen had the desquamative affection, of which thirteen belonged to the class in which there was no doubt of syphilis, one was doubtful, and the other showed no evidence of lues. As a *résumé* we would say that of the forty-six cases observed by Parrot, syphilis could be established by him in forty-one cases. The next thing to be taken into consideration is that Parrot was in the habit of considering manifestations as syphilitic which have not since

been accepted as such. So that we may safely reduce the number forty-one somewhat. Let us grant, however, that all of the forty-one cases of the desquamative affection had syphilis, what reasons are given that the affection is a syphilitic one? None, except that the affection occurred in syphilis. The disease lasts an indefinite length of time it is found, according to Parrot himself, in patients showing no other manifestations of syphilis; pathological investigations do not show any specific lesions; and, lastly, still according to Parrot, treatment has no effect upon the disease. If we now consult the experience of other writers we will find that there is no one who can subscribe to the syphilitic origin of the "wandering rash." Parrot has then been misled by the observation of a coincidence into establishing the relation of cause and effect, and the term "desquamative syphilide of the tongue" must be stricken from our list of diseases, certainly, as applied to the "geographic tongue" or "wandering rash." I do not think it would be rash to state that in the three hundred and twenty-eight cases of hereditary syphilis observed by Parrot a greater number than fifteen would have been found that had stomatitis mycosa, and yet no one would think of considering thrush as a syphilitic manifestation. Syphilis, then, plays no rôle in the production of this disease; the question that arises is as to its etiology. As has been stated, we do not know what are the causes of the "wandering rash." The most commonly accepted view that it is due to some general disturbance of health or to a stomachic trouble is fallacious. This will be proved by long-continued observation of any individual case as well as by therapeutic attempts. No one ever succeeds in curing this form of tongue either by tonics or by diet. It is doing the patient an injustice to restrict his mode of life in the vain attempt to cure an affection which is harmless and, as far as we know, incurable. There are no symptoms attached to the affection; some authors speak of an itching sensation, but this certainly must be excep-

tional. In the great majority of cases the diagnosis is an accident, made when the tongue is examined for other reasons. I have observed four cases in which heredity seemed to play a rôle: two children in two families, the mother in one and the father in the other being brother and sister. The cases are, however, not of sufficient frequency for any one observer to work out any law concerning their origin. It has seemed to me that we are dealing with a process of epithelium formation which is natural to those individuals affected, and only to be considered abnormal in that it does not agree with the production of epithelium in the great majority of human beings. The slight changes which have been found under the microscope could be just as readily explained by considering them the effect and not the cause of the trouble.

Syphilitic teeth.—The changes in teeth due to syphilis have received considerable attention since they were first described by Mr. Jonathan Hutchinson, and quite a literature has grown up around this one subject. The question is one which has not been conclusively settled, but, nevertheless, the changes which were originally described, and concerning which Mr. Hutchinson has written so often and so well, have been accepted by so many as characteristic of syphilis that it will require the thorough record of a great number of well-observed cases to disestablish this view. On the other hand, a great many changes in the teeth have been noticed and been ascribed to syphilis which, certainly, are produced by a great many other causes. Thus Parrot (*loc. cit.*) divides the “*odontopathie atrophique*,” as he calls it, into five kinds: the cup-shaped, the sulciformed, the cuspidated, the notched, and the axe-shaped. All these are, according to this author, distinctly due to syphilis. He admits that rickets may produce one or the other form, but, according to him, “rickets is nothing more than the last manifestation of syphilis upon the osseous system.” Without going into details in the discussion of this whole subject, it has reduced itself to the

following: Mr. Hutchinson has proven most conclusively that the forms of change which he has noted in his cases were due to syphilis. He has been enabled, by looking at what he considers as characteristic, to read the history of his patient, of his parents, and sometimes of his grandparents. Again, as a result of his deduction, he has been enabled to arrive at therapeutic conclusions which have invariably proved the correctness of his clinical evidence. There is one important link missing in the chain of evidence brought forward by Mr. Hutchinson: no one has ever seen a lesion in the tooth-sac which would prove the correctness of the connection of syphilis with these peculiarly-shaped teeth. One single case, in which a pathological change could be attributed to syphilis, would establish the view most positively. It must be remembered that a great many excellent observers (Nicati, Albrecht, Bouchut, Grünfeld, etc.) do not recognize these syphilitic teeth as pathognomonic. Again, it must be admitted that it is a very simple matter to call a child syphilitic because it has notched incisors. It does not prove that because the child has notched teeth, and either or both of its parents have had syphilis, that the syphilis is the cause of the notched teeth. If this combination of circumstances is found in a great many cases it might be looked upon as presumptive, but by no means as conclusive, evidence. The subject of tuberculosis might be adduced as an illustration; by the general consensus of professional opinion, phthisis was looked upon as an hereditary disease. No one, to-day, looks upon heredity as of any more value than a predisposing cause, either direct or indirect. Bouchut claims that any diathesis or cachexia may produce these peculiar teeth, and adds that he has seen cases in which neither parent had syphilis. ("Clinique de l'Hôpital des Enfants Malades," p. 357, 1884.) The case that he reports, due to an epistaxis and followed by illness during the whole of infancy, seems to settle the whole question. But the great

objection, which unfortunately can be raised in all the cases reported as non-confirmatory of Mr. Hutchinson, is that the history does not seem to have been examined as carefully as the subject would warrant. Bouchut's view, if correct, broadens the etiology, but his evidence is, if anything, weaker than Hutchinson's; nor can it be said that the former has added one iota to our conception of the whole process. It is possible that syphilis, like heredity in tuberculosis, is a predisposing cause for the formation of these teeth. We know perfectly well that certain diseases will leave their marks upon the teeth, because we see that those teeth which make their appearance after certain illnesses are deformed. If one lesion, why not another? If an erosion, why not a notch?

Mr. Hutchinson has met this question with the answer, that syphilis produces only certain changes, which is eminently proper, but which may be overturned by subsequent observation. On the other hand, it is exceedingly difficult, perhaps impossible, to prove that syphilis has produced certain well-defined lesions.

The whole subject has not been conclusively settled, but from all indications certain lesions of the teeth must be looked upon as very strong evidence of the existence of hereditary syphilis. In the presence of these changes the patient or his parents would have to have a very decisive history proving the non-existence of syphilis to make it positive that hereditary syphilis was not at the bottom of the lesions. In the majority of instances, however, we have additional evidences which help us in our diagnosis, so that we are not frequently called upon to rely implicitly upon one sign.

On account of the ready acceptance of Hutchinson's teeth by the profession, a great many other forms of deformed teeth have been described as due to hereditary syphilis. It may be just as well to state, in the present connection, that for these other forms the proof is by no means as convincing as that

offered by Mr. Hutchinson. As will be seen, the only evidence given is that a certain author has described a certain form of tooth as characteristic for syphilis. While the author may be a most competent observer, such a statement is absolutely valueless when it is not enforced by a careful analytical report of cases.

Hutchinson's teeth.—The "test teeth" are the two upper, central, permanent incisors. In addition to these, however, there is a symmetry of deformity which is also supposed to be characteristic of syphilis. The "test teeth" are "dwarfed and notched." They are somewhat or very much smaller than their neighbors; they are exactly alike in shape and size, and are affected alike in every respect. Their size depends upon the intensity of the affection as well as upon the time when manifestations of syphilis have occurred. If a patient shows the manifestations of hereditary syphilis after the tenth or twelfth year of life it is impossible to find markings upon the "test teeth." It is questionable whether hereditary syphilis remains latent for so great a length of time; but this claim is made for it by authors. ("Syphilis hereditaria tarda.") The cause for the deformity is expressed as follows by Mr. Hutchinson (*London Hospital Report*, II. p. 148): "The teeth . . . are developed in connection with the mucous membrane; are parts of a dermo-skeleton. Their pulps suffer accordingly in an inflammation of the structure, and hence the malformations which are produced." If this be the case, the greater the inflammation the greater the deformity.

In hereditary syphilis the two central incisors are usually separated from each other by an abnormally great space. The notch, which is characteristic, is found upon each incisor, not mathematically alike in each tooth, but in almost the same place. It is vertical, sometimes broader at the grinding edge than above, and deeper, extending into the substance of the tooth for some distance like a slit, limited at both ends by an

outline whose convexity is in the same direction. Most commonly it is nothing more than a semicircular notch of varying depth. In addition to this notch, the lateral outlines of the teeth are decidedly convex instead of being straight. Next to the central incisors, the two lateral are mostly deformed; they are dwarfed and have convex outlines. The canines, too, may be dwarfed, also equally. The rule is absolute symmetry of deformity, but this rule is subject to exceptions; everything else being equal, symmetry speaks for syphilitic deformity, non-symmetry, for malformations from other causes.

Another form described by Mr. Hutchinson is the "screw-driver teeth." Of these he says (*loc. cit.*), "Among cases in which the teeth are peculiar without being pathognomonic, we very frequently meet with those in which the central incisors of the upper jaw are narrowed but not notched." This narrowing extends from the crown towards the edge, is symmetrical in the upper permanent incisors, and is combined with dwarfing. In the same article we find "some of the screw-driver teeth are almost as useful to the trained observer as are the notched ones." However, they are admitted as not pathognomonic, and I have certainly seen cases in which there seemed to be no evidence of hereditary syphilis.

We have already quoted Parrot. Fournier makes at least three other forms of syphilitic teeth in addition to those of Parrot. From a simple loss of enamel in depressed spots to an absence of part of the tooth, all are alike produced by syphilis. Not one of the forms is characteristic for syphilis, but they are all probably produced by reduction of general health, disturbances in nutrition of the tooth while developing, or hereditary tendencies of varied sorts.

As far as the effect of syphilis on the time of the eruption of the teeth is concerned, this must vary with every individual case. If the syphilitic child becomes affected with rachitis,—and the rule that all syphilitic children become rachitic does

not hold good in this country,—dentition will be interrupted or retarded. If the patient has a severe attack of infantile syphilis, the teeth will be slow in coming, just as if the general nutrition of the child had become affected by any other deep constitutional disturbance. If, on the other hand, the syphilitic invasion has been slight, teething may go on in the normal way. I have now under my observation a child in whom the history has been as follows: Father syphilitic; married against advice. First pregnancy followed by abortion; second, by still-born child at term; third, by child with papulo-pustular syphilis; fourth, by healthy child, developing macular syphilide six weeks after birth. Vigorous treatment of father, mother, and children by mercurials. The last baby, now thirteen months old, has nine teeth. The whole process of teething has gone on in a normal way; the first two lower incisors appearing about the fifth month, followed in four months by the upper incisors.

It is possible that the permanent teeth are affected according to the same rules as the deciduous; but of this the dentists are better judges, as physicians are more concerned in the first than the second teeth. It is not unreasonable to suppose that the more intense the infection the better marked will be the evidences of syphilis. Yet, as far as the teeth are concerned, this is not borne out by experience. Various observers state that, in their experience, Hutchinson's teeth have been very rarely observed, and some go so far as to claim that, in their own countries, this form of malformation is exceedingly rare. All authors admit the fact that very many syphilitic children do not have malformed teeth, and every one, I think, is able to verify this statement. Again, there seems to be no relation between the intense forms and the appearance of this symptom; sometimes a mild case is followed by Hutchinson's teeth, at others a severe one. The conclusion which must be arrived at is that Hutchinson's teeth depend upon the inva-

sion of the mouth, depending upon the intensity of local manifestations, and not upon the intensity of the general affection.

Treatment of Stomatitis Syphilitica.—This is not the place to speak of the general constitutional treatment of syphilis. As in all forms of syphilis, so in syphilis of the mouth, constitutional treatment plays a most important rôle. For those forms of stomatitis syphilitica which are apt to produce deformity, or rapid destruction of tissue, that kind of general treatment which produces its effect in the shortest possible time is indicated. For those other forms which do not produce any great amount of local damage, or which are only minor complications of a great general disturbance, that treatment is to be used which best fits the type of general affection.

There is hardly any manifestation of syphilis which can be so readily affected by local treatment as the syphilitides of the mouth. Again, there is no other form of syphilis which is so easily aggravated by local irritations. In view of the latter statement, prophylaxis becomes a very important factor in all of these cases.

Although salivation is very rare in children, yet the careful physician watches the mouth when he gives mercurials to children. In the treatment of syphilis by long-continued use of mercurials, all those prophylactic measures used against salivation in adults should be applied to children, especially when there is any evidence of beginning mouth-trouble. Some of these ought to be used in all cases, whether mouth-trouble be threatened or not. If the child has teeth, they ought to be kept scrupulously clean, either by a tooth-brush or by other means. When there are no teeth, an antiseptic mouth-wash should be used,—salicylate of soda, permanganate of potassium, listerine, or lukewarm water, which is as good as anything else when employed in sufficient quantities. The principle upon which all prophylactic measures are based is cleanliness. Physicians very frequently consider themselves as acting anti-

septically when they use a solution of one or the other anti-septic remedy. Combined with this there may be septic fingers, septic instruments, and gross negligence in overlooking those parts which require treatment most. The remedy used is simply for the purpose of easing the conscience, and when bad results follow, physicians are astonished, and attribute the results to some peculiarity of the patient instead of to themselves. Absolute cleanliness is worth more than all antiseptics in the treatment of diseases of the mouth ; antiseptics without cleanliness are worse than useless.

All irritations within the mouth must be removed. Especial reference is made here to the teeth. If they are decayed, they must be attended to, either by filling or extraction ; if they are very sharp, they must be filed off. The latter is of great importance, as many a syphilitic ulcer has been kept up in the mouth by the constant irritation of a sharp tooth. This is rare in childhood, although it has been known to occur.

The sovereign local remedy for all syphilitic ulcers, erosions, or losses of substance in the mouth, is nitrate of silver, applied in the manner described in connection with stomatitis catarrhalis. Corrosive sublimate is especially indicated when there is very much infiltration in addition to these ulcers. It can be used either in dilute or concentrated solutions, and by using glycerin the concentration can be made very great (almost equal parts). The dilute solutions (0.2-1 per cent.) can be used with a swab to the inner surface of the mouth. If the child be old enough, the weakest solution can be sparingly used as a mouth-wash. In infants all the weak solutions ought to be applied from two to four times daily. The very strong solutions (as high as twelve per cent.) are to be used externally ; only in cases of excoriations or rhagades that resist the nitrate of silver or weak corrosive sublimate solutions. These strong solutions are caustic in their effect, are followed by pain, and, not infrequently, by more or less inflammation. They

must be handled very cautiously ; their indication is very limited, and they need not be applied oftener than two or three times. Where children are old enough, emplastrum hydrargyri, softened with lanolin, frequently gives better results than anything else in syphilitic cracks of the corners of the mouth. The best mouth-wash, in syphilitic cases, is a solution of chlorate of potassium. There is no remedy which counteracts the tendency to stomatitis mercurialis better than this, and none which acts more promptly, as has already been pointed out in connection with stomatitis ulcerosa. It sometimes becomes necessary to use an astringent as a local remedy. Any substance containing tannic acid will fulfil the indication perfectly ; either tannin itself—although objectionable in young children on account of its taste—or *tinctura ratanhiæ*, *tinctura catechu*,—the latter being least objectionable.

STOMATITIS PRODUCED BY LEPTOTHRIX BUCCALIS.

This is an extremely rare affliction in children. From the investigations of Miller ("The Micro-Organisms of the Mouth," 1890) we are led to the conclusion that *leptothrix* is not a pathogenic organism. He has described at least four varieties, all of them uncultivable and of unknown pathogenesis. His position, and undoubtedly the true one under present conditions, is "morphologically as well as physiologically considered, *leptothrix buccalis* has been regarded as a veritable wonder. It has been said to perforate and split up teeth, its elements to cause all kinds of diseases in the oral cavity, to penetrate into the lungs, the stomach, and other parts of the body, and everywhere to manifest a destructive influence. As absolutely nothing was known concerning the biology and pathogenesis of this organism, all sorts of wonderful properties were ascribed to it. It is, therefore, high time to banish this confusing name from bacteriological writings."

Clinically speaking, a lower form of life that has been called

leptothrix forms membranes within the mouth, about the teeth, the circumvallate papillæ of the tongue, and especially upon the tonsils. These membranes are all characterized by their dark, grayish-black color and their firm adherence to the soil upon which they grow. These evidences are sufficient, as a rule, to establish a diagnosis; an examination under the microscope will show the absence of *saccharomyces albicans*, or any of its organs, and the differential diagnosis will, therefore, be very simple. The symptoms that are produced are very slight, and this may account for the great rarity of the disease in children. It is only when the membrane appears upon the tonsils that adults complain, and in my own experience a case of stomatitis leptotrichia has never occurred in children.

VIII.

DENTITION.

No subject in pediatrics possesses so large and varied a literature as that of teething. Upon no subject do we find more diversity of opinion than upon teething. It is, therefore, very natural that great importance should be attached to its discussion, which ought to be carried on in a thoroughly objective manner. The latter, unfortunately, is an exceedingly difficult matter, for more than one reason. Nearly all the medical superstitions of childhood pertain to teething. Social position, education, culture, do not seem to eradicate them; a string of amber beads, a mouse's tooth, or a bag containing some odoriferous compound may be found in the mansion as well as the tenement-house. It is not an easy matter for the physician to stand by patiently and have his skill counteracted by an amulet. Moreover, the idea of the causation of disease by teething has taken such deep roots in the minds of people that diseases easy of treatment end fatally because they are either overlooked or attributed to the process of teething. It is not difficult to answer the question, "Who is to blame?" If a cursory glance be taken at the literature of the subject, authors will be found—and good ones, too, both old and modern—who do not hesitate to ascribe any form of disease to teething. The more improbable the connection between teeth and disease, the more delight does the average author find in stating that the affection is due to teething; or, if he be of a speculative turn of mind, the more ingenious will be his method of tracing cause and effect. It would be an interesting though useless task to collect all the diseases that have been evolved from teething. Then there are those who, writing for and not from experience, copy what has been said by others, accepting facts badly collected and not thoroughly verified;

these are the ones that go to make up the number who assist in the errors that are being made daily, for they are authors, and an author is supposed to be an authority. Hebra, the great dermatologist, who detested rushing into print, would say to the person who entertained a new view concerning eczema, "When you have seen ten thousand cases of eczema come and talk to me." It is not necessary to have seen ten thousand children teething,—in fact, not one-fifth the number; but it would be conducive to the welfare of humanity if physicians would take the trouble to observe teething children more and believe less in what has been written on the subject. Almost daily do we see errors in diagnosis occur because of the doctrines that have been and are being taught on the subject of teething. This statement can be substantiated by any number of cases in the experience of the observing physician.

It is but natural to ask for patience and quiet, impartial judgment in the discussion of this subject, when one sees the errors that are committed as the result of this kind of teaching. Within a short space of time I have seen the gums lanced in children suffering from pneumonia, measles, and tubercular meningitis; in each case the diagnosis of teething had been made, in each case the child died, and in each case both the physician as well as the surroundings were settled at the bedside with the stoicism of followers of Mohammed.

We deal with two processes: one, normal dentition, which is admitted to exist even by the most rabid adherents of the teething doctrine (only in about twenty per cent. of all children, however); the other, abnormal dentition, or *dentitio difficilis*. Before going on to the further description of the two processes, it will be both interesting and profitable to look into the history of the subject.

The late L. Fleischmann, of Vienna, published a small volume in 1877 ("Klinik d. Pediatrik"), in which a complete history of the subject will be found, and from which the fol-

lowing abstract is made in part. Fleischmann was one of the most thorough authors who has ever written upon pediatrics, and his early death left a great void in the ranks of active workers in diseases of children.

We again begin with Hippocrates (400 B.C.). In the apocryphal work "De Dentione," we find that children teethe more easily when they have loose bowels, and better in winter than in summer; that those having a cough teethe late and lose flesh. In his "Aphorisms," which are considered genuine (Sect. iii.), the statement is made that teething children suffer with itching of the gums, fever, convulsions, and diarrhoea, especially when the eye-teeth come through. Fat children and those that have a tendency to constipation suffer most. He knew that the teeth are formed during foetal life, and advised that the children be allowed to bite upon hard substances during teething.

Aristotle (384-322 B.C.) had erroneous notions concerning teeth: that men have more teeth than women; that the teeth continue to grow during the whole lifetime of the individual, and that repeated teething occurs in strong people.

Galen (131-203 A.D.) tells us that the teeth act as foreign bodies during eruption, and that they produce all possible bad symptoms, because they cannot be encapsulated like foreign bodies. Some of his pupils believed the most wonderful things: the gums should be rubbed with dog's milk, or with the brain of a rabbit, in order to cause the teeth to come through more rapidly; here, too, we find the first mention of an amulet's power against the pains of teething, "Veteris cochlearum cornu pelliculæ illigatum pro amuleto appende." The first tooth was considered an epiphysis of the permanent tooth in order to explain Aristotle's erroneous notions. Oribasius (326-403 A.D.), an indirect follower of Galen, prefers Cyprian butter, or oil of lilies, to the brain of a rabbit.

Aetius, at last, developed the whole subject of diseases of

dentition. Irritation from the tooth produces inflammation which may extend to the ear, the nose, the eyes, the stomach, may produce fever, etc. The child must not be allowed to become constipated, otherwise the inflammation will extend upward. Oil must be used to rub the gums, but hard substances must not be given to the child to bite upon, as they harden the gums ; amulets (*e.g.*, the tooth of a viper encased in silver) are of great value.

Paul of *Ægina* (625–690) states that convulsions are apt to occur during teething : these he counteracts by washing the child with a decoction of heliotrope ; he cures diarrhoea by placing a spice-bag with rose-seeds upon the bowels (it is pleasant to meet some of our old friends twelve hundred years back) ; the itching of the gums is diminished by the use of old herrings.

Rhazes (860–932), in his work “*De Ægritudinibus Puerorum*,” writes as follows : “ Prorruption is easy and is accompanied by little pain when the teeth come through rapidly, but they are not strong, and, *vice versa*, when they grow slowly the pain is greater but the teeth are stronger.” Teething is easier in spring than in winter ; in summer teething is painless, but abscesses of the cheeks and gums, as well as itching of the ears, are more common ; hemorrhages, fever, diarrhoea, or constipation may also be present. He makes no mention of convulsions.

Avicenna adds nothing original to the work of his predecessors.

Vesalius (1514–1564) was the first to practise incision of the gum in case of a wisdom-tooth.

Eustachius was the first to controvert the views of Aristotle, Galen, and Vesalins, and to verify the statement of Hippocrates,—*i.e.*, that the teeth are already formed in the foetus. He also opposes the view that the eye-teeth have anything to do with the eyes.

Ambroise Paré (1510–1599) first advised lancing of the gums in difficult teething. He reports that those teeth that

had been lanced in a child dying from difficult teething came through even after the death of the child.

After this period the most curious theories are to be found in the literature of teething. Scarification is almost universally recommended; amulets increase in number, and are divided into classes according to their supposed strength.

This sort of medication continues through the seventeenth century; in the eighteenth century the amulets are dispensed with; but the number of diseases and symptoms due to teething is gradually increasing. John Hunter (1772,—*The Works of J. Hunter*, 1835) gives the following list: “Diarrhoea, costiveness, loss of appetite, eruptions on the skin, especially on the face and scalp, cough, shortness of breath, with a kind of convulsed respiration, and similar to that observed in whooping-cough, spasms of particular parts, either by intervals or continued, an increased, and sometimes decreased, secretion of urine, a discharge of matter from the penis, with difficulty in micturition, resembling symptoms of gonorrhœa in its violent form. The lymphatic glands are apt to swell at this time; if the child has a strong tendency to serofula, this irritation will promote the disease. There may be many other symptoms with which we are not at all acquainted, the patients, in general, not being able to express their feelings.” For treatment, cutting the gums is “the only method of cure.” “It often happens, particularly when the operation is performed early in the disease, that the gum will reunite over the teeth; in which case the same symptoms will be produced, and they must be removed by the same method.” He has lanced the same teeth ten times, giving relief each time, but followed by a relapse.

Jacob Plenk (1779) adds the following list to those diseases already mentioned: Gutta rosacea, deafness, amaurosis, swelling of the knees, paralysis, and lameness in one or both legs, suppuration, and dry gangrene.

Rosen von Rosenstein not only believes that any ill can

result from teething, but thinks that every child ought to be prepared for teething as soon as it reaches the age of three months; the gums should be rubbed with the finger every day in order that they may become thin enough for the teeth to come through without giving pain. If this has not been done, he recommends one of a thousand and one remedies to soften the gum as well as giving relief to pain by means of venesection, leeches, and scarification.

Girtanner (1796) adds a few more diseases to the long list,—increase in the flow of bile, nausea, stomach cramps, fainting, and epilepsy.

Armstrong (1786) is the first who dares to lift up his voice against the views that have been expressed. He does not believe that all cases dying about the time of dentition die from teething, and warns against the too free use of the gum lancet, having seen the scarification of the gums followed by fatal issues.

Beginning with this, we first find Wiedmann (1800) opposing the views expressed by the older authors with great firmness and with all the logic and force that followed the knowledge of physiology. He ends his long article as follows: "It is to be hoped that, in the future, dentition will be called up only when it would be impossible to comfort the relatives with the impotence to designate the true nature of the disease or to quickly calm the laity."

From this time the two opposing camps have been formed: in the one, those believing in the possibility of production of all kinds of disease by teething; in the other, those believing in the production of teeth, and teeth only, with occasional bad symptoms by this same process. On both sides there are champions worthy of the cause: Barthez and Rilliet, West, Bednar, Steiner, Vogel, and a great many others, for the diseases produced by dentition; Politzer, Bouchut, Fleischmann, and, in our country, Jacobi especially, against this view. Jacobi has done more than any one, probably, to place the

whole subject of dentition upon its proper level (1862); and the thoroughness and analytical acumen displayed by him in his argument are such that it requires but close following to be thoroughly convinced of the truth of his deductions. In order to be able to make deductions, it is necessary to take a cursory view of the whole process of teething as it goes on normally. It will be readily understood that a detailed description, histological and biological, would be entirely out of place here; it is our object to view the conditions from the aspect of topographical anatomy, so that reference to it can be had in the following discussion.

The statement that the greater part of the process of teething is accomplished before the child is born will, I hope, be thoroughly verified by what follows. About the seventh week of intra-uterine life a ridge is formed within the mouth, caused by a thickening of the oral epithelium, and at the same time a dipping down of this same epithelium into the embryonic tissue which goes to make up the jaw. The epithelium which dips down is called the enamel germ; by growth and extension it shortly becomes converted into a flask-shaped outline, resting upon the embryonic tissue and lining a cavity. Partitions are formed in this continuous, irregularly-shaped depression, so that there are differentiated ten small bodies in each jaw. At the same time a papilla forms from below which grows upward, and, in a short time, develops to form a complete mould for the enamel germ which comes to rest upon it, and forms its lateral and upper boundaries. This papilla is highly vascular, and is called the dentine germ. While this is going on the connective tissue around the primitive tooth differentiates itself to form an investing membrane for the tooth, called the dental sac. Osseous tissue is being formed all the time in the partitions between the primitive teeth, and finally we have the alveolus formed around the tooth-sac, "at first with wide openings which afterwards are narrowed, but so as to allow

the contained sacs to cohere with the gum along the border of the jaw" (Schaefer). We have now arrived at the time of birth, and it will be necessary to see what has become of the various tissues we have seen developing. The enamel germ, with its two layers, forms the internal lining of the dental sac and the enamel itself. The dentine germ has formed the dentine, the pulp-cavity of the tooth, and the beginnings of the fangs. The dental sac, enclosing the tooth, has itself two layers, an outer vascular connective-tissue layer and the inner one, part of the enamel organ, going to form the so-called cuticula. The alveolus has been formed by bony deposit with its opening quite wide (Fleischmann), wider than is necessary for the crown of the tooth to pass through. As far as the development of the whole jaw is concerned, this must be looked upon as disproportionately great for the small number of teeth, so that there is more than enough room for all the tooth-sacs and no crowding, the one upon the other. The permanent teeth are formed, practically, in the same manner as the temporary, except that there the enamel germ is formed from the enamel germ of the temporary teeth, in the shape of a small sac which goes to its development in the same way as it did in the temporary tooth.

At the time of birth, then, we would have an individual tooth—let us take the lower central incisor—with the following topographical relations: Above, we find the tooth-sac, the submucous connective tissue, and the mucous membrane; on either side we find the tooth-sac and bony substance. The tissues which interest us most are those that separate the tooth from the oral cavity. The tooth-sac is very thin, and offers very little resistance to the upward or downward growing tooth; the same can be said for the mucous membrane. The submucous connective tissue is comparatively thick, and is the only substance that can be taken into consideration as opposing the developing tooth, as the alveolar cavity has been shown to

have a sufficiently large outlet. The force that causes the teeth to come through the gums is the calcification of the fangs. As a result of this calcification the tooth is elongated, and, enclosed as it is, room has to be made for this elongation. It is not surprising to find that the tooth is forced in the direction of least resistance, and, being everywhere surrounded by cartilaginous and bony tissue except in the direction of the mouth, it naturally begins to move in this direction. We have seen, furthermore, that the resisting medium is the sub-mucous connective tissue. Let us go on and find how this resistance is overcome.

The force at work is long-continued slight pressure, and this causes atrophy of the mucous membrane. This begins to work at different periods, depending upon the development of the teeth ; at birth it begins in the lower incisors, shortly after in the upper incisors, so that with the completion of the third month of life the molars are already beginning to have their roots calcified (Pierce, "American System of Dentistry"). The question, "When does dentition begin?" must be answered by "at birth," provided we are satisfied with considering the beginning of dentition as applied to the beginning of pressure upon the soft tissues. This, as will be seen, is perfectly logical, as nearly all the symptoms attributed to teething are supposed to be due to pressure. On account of the innumerable symptoms that have been pointed out it is impossible to state when teething does begin ; it is not uncommon to find the eruption of teeth within a few days prophesied by wise persons who rely upon certain symptoms, and the teeth not forthcoming within as many months. It becomes a question then, When does teething begin ? when the teeth begin to break through into the soft tissues, as answered above, or when the teeth make their appearance in the mouth ? The latter is the one that can be used to greatest advantage for more reasons than one, not the least important being that the time between

the appearance of the first and last teeth is longer and that we have some visual testimony of the process. It must be added, however, that very few would be willing to place the beginning of teething at the time of the appearance of the teeth, and, to a limited extent, they are correct, as certain symptoms frequently precede the eruption of a tooth; these are so illy defined, so varied, and the time of their manifestation is so various, that no reliance can be placed upon them.

I have collected a list of modern authors in tabular form in order to show how widely they differ on the subject of normal teething, and to bring out another point which will be discussed hereafter. It is a strange commentary upon the existence of accurate knowledge that the views of authors differ so widely upon a subject so common as teething. It has caused not a little difficulty to arrange these views under the three headings that follow, and a close examination of the tables will show that some of the authors quoted cannot, strictly speaking, be brought into a class with any others but themselves. In this connection, it would be useless to examine into the causes of this diversity of opinion. Not the least important factor, however, is that most physicians have received their first information on the subject from the anatomists. The anatomists, however, would manifestly be the last persons to go to for information regarding a process that goes on in childhood, their observation being confined almost exclusively to the adult. In witness to the truth of this statement it is only necessary to recall the fact that anatomical research, as applied to infancy and childhood, is only beginning. Furthermore, where the anatomists have examined into this question,—and some of them have done it extremely well,—they have had for material children who died of some disease that has probably affected the time of the appearance of the teeth. Again, it is certainly necessary, in order to arrive at proper conclusions in regard to a physiological process, to examine into it while it is going on, and that is during life. Results obtained

in any other way must, of necessity, be erroneous. The list is far from complete or exhaustive, but will, I think, illustrate what is to be shown. Three orders of appearance have been put down for normal teething: 1. The appearance of the teeth in pairs,—*i.e.*, as far as the incisors are concerned. 2. The appearance of the first two incisors, then all the others, and then the molars. 3. The appearance of the first lower incisor, then the four upper incisors, then the first molars, and with them the last lower incisor.

The first teeth are put down, by most authors, as being the two lower central incisors, although a goodly number of authors will be found that believe in the prorruption of the lateral incisors as the first. My own impression is that, in the great majority of cases, the third method of teething is the normal one. The authors who claim that normal teething goes on in the first manner described above are as follows:

Two lower Central Incisors.	Two upper Central Incisors.	Two lower Lateral Incisors.	Two upper Lateral Incisors.	Four First Molars.	Four Canines.	Four Second Molars.	
6-7 months.	4-6 weeks. +	4-6 weeks. +	4-6 weeks. +			End of 2d year.	Hyrtl, 1873.
7 months.	2 weeks. +	4-6 weeks. +	4-6 weeks. +	12-14 months.	16-20 months.	20-30 months.	Tanner and Meadows, 1871.
7-8 months.	2 weeks. +	4-6 weeks. +	4-6 weeks. +	12-14 months.	3-4 months. +	24-30 months.	Day, 1881.
3-10 months.	9-16 months.	13-17 months.	13-17 months.	16-21 months.	16-25 months.	23-36 months.	Bagiusky, 1887.
6-12 months.		10 months.	11 months.	12-16 months.	17-20 months.	20-24 months.	Fleischmann, 1877.
7 months.	6 weeks-2 months. +	In a short time. +	In a short time. +	3-4 months. +	3-4 months. +	24-30 months.	West, 1874.
7-9 months.				12 months.	16-20 months.	Second year.	Eustace Smith, 1884.
6-7 months.	At end of 12 months.	At end of 12 months.	At end of 12 months.	12-16 months.	16-24 months.	24-30 months.	J. Lewis Smith, 1881.
7-9 months.	Several weeks. +		End of first year.	15-18 months.	18-20 months.	20-24 months.	Henoeh.

Those who claim that normal teething goes on in the second manner are as follows :

Two lower Lateral Incisors.	Six Incisors.	Four First Molars.	Canines.	Four Second Molars.	
6-9 months.	2 weeks-3 months.+	First half 2d year.	Second half 2d year.	Third year.	Schnitzer and Wolf, 1849.
5-8 months. (4 central.)	7-10 months (4 lateral.)	12-16 months.	14-20 months.	18-36 months.	Semple, 1884.
6-7 months.	7-9 months.		Beginning of 2d year.	End of Second year.	Henke, 1830.

To which may be added Meissner (1844), who makes teething begin at seven months and end with the second year of life, and Wendt (1835), beginning with the 20th-28th week after birth and ending the middle of the second year.

Authors who believe the third method to be the rule are as follows :

Two lower Central Incisors.	Four Upper Incisors.	Four First Molars and two lower Lateral Incisors.	Four Canines.	Four Second Molars.	
4-7 months.	8-10 months.	12-14 months.	18-20 months.	28-34 months.	Gerhardt, 1874.
4-7 months.	8-10 months.	12-15 months.	18-24 months.	20-30 months.	Starr, 1886.
5-7 months.	8-10 months.	12-15 months.	18-24 months.	30-36 months.	Hüttenbrenner, 1888.
4-7 months.	8-10 months.	12-15 months.	18-24 months.	20-30 months.	Vogel, 1874.
7-9 months.	8-10 months.	12-15 months.	18-20 months.	20-30 months.	Jacobi, 1887.
6-8 months.	8-10 months.	12-14 months.	16-20 months.	20-36 months.	Dorning, 1889.
2-15 months.	11-12 months.	17-18 months.	2 years.	30 months.	Barthez and Rilliet, 1861.

As has been seen by these tables, a wide difference exists, not only as to the order of dentition, but also as to the times of the eruption of the different groups. The earliest time

given for the appearance of the first tooth is two months, the latest, fifteen months, although the same authors (Barthez and Rilliet) state that normal teething may begin at any time during the first two years of life. The termination of teething is put down, at its earliest, at eighteen months, and at its latest at thirty-six months; concerning the latter dates there seems to be more uniformity of opinion than concerning the beginning of dentition.

The questions to be discussed are: Who is right among these observers? and how are these observations, apparently irreconcilable, to be explained?

A great many authors have put down the two limits instead of the average time of their observations, so that we can readily understand the statements of Barthez and Rilliet. Again, the time of eruption of the first teeth depends entirely upon certain conditions of the child and its surroundings. Nationality, therefore heredity, plays a most important *rôle*: French children teethe early; English, Russian, German, and Italian children one to two months later on an average, and Hungarian children still later. In this country, on account of the presence of so many nationalities and their mixture, we naturally find that the average time of teething depends largely upon the material taken for the calculation. It is my own impression that the average child has its first two teeth with the completion of the seventh month, and this seems to accord with the statements of most American authors (Jacobi, Dorning, J. Lewis Smith), Starr alone having retained the four to seven months for the first two teeth, as put down by Vogel and Gerhardt, which must surely represent exceptions and not the rule.

It is stated that climate has an effect upon the time of teething (Fleischmann), and this can be accepted, as the explanation would be found in the effect of climate upon the general constitution. The latter, after all, is the predomi-

nating cause for the early or late eruption of teeth, everything else being equal. This has been proven very frequently, although, possibly, in a given individual case it may not be true. The development of the teeth goes on equally with the general development; a child well developed for its age will usually have its teeth early and regularly, and the converse of this holds true.

There are certain diseases which retard the eruption of teeth; but beyond this, there are children that teethe late without any appreciable cause. It is going too far to state, as Rehn does, that every case of delayed dentition is due to rickets. The observation is frequently made, and I am prepared to verify it fully, that there are certain families whose children teethe late, and yet these children are in good condition in every respect. The time of eruption depends for its physical basis upon: first, the distance that the tooth has to go through from the dental sac to the mouth; secondly, the time when the calcification of the fang begins; and, thirdly, the condition of the organs from which the tooth develops. If the distance be great, it can be overbalanced by an early and great deposit of calcareous matter in the tooth; a tooth which is quite superficial might be long in coming through if the deposit is not sufficiently early or great. Lack of development in the rudimentary organs could be compensated for, partly, by an early and great calcification; but it is manifest that this could only be to a certain degree, and in some instances no compensation could take place in any manner. Now, the deposit of calcareous matter is a process depending mainly upon a proper supply of raw material in proper form, and this, again, is due principally to general causes, and not to those acting in a purely local way. There are those effects upon the embryonic structures which are never overcome (*syphilis*); again, there are those which leave structural changes (*diseases of the foetus*); and, finally, those

that produce a great retardation of eruption (rickets, fevers during infancy, etc.).

As far as the order of eruption is concerned, it is not difficult to reconcile the various methods that have been described. The fact remains that, as in the case of normal labor, we must make a compromise in order to state which is the normal method of teething. This can be done only, as it has been done in obstetrics, by a carefully-conducted statistical research, which shall include a great number of cases. In this way we would be able to establish one way of normal teething instead of the three put down by authors. As it is, we must admit that normal dentition may go on in any one of the three ways, and that the differing views mean only that one author has observed his way most frequently. It certainly seems strange that we still find authors, including one on dentistry, who find the canine teeth appearing before the molars, and it is very difficult to explain these statements, if we consider them based upon observation on the part of the authors themselves.

It is more than likely that the latter is not the case. The mistake, on the part of a dentist, is more than excusable, as dentists have their principal dealings with adults. It is certainly gratifying to know that those dentists who are abreast with the times give as much attention to the teeth of children as to those of adults.

The reason why certain teeth appear before others depends upon the explanation given before. The more perfect the embryonic structures, the nearer the surface of the mouth, and the smaller the tooth, the sooner will it come to the surface. These are the factors that cause the incisors to appear first, to be followed by the molars, and then by the canines. In the latter, it is the calcification of the fang that causes its late appearance; it is not due to some far-fetched metaphysical reason, as claimed by some authors, but simply a question of lime-salts.

We must look to the same causes for premature dentition as we do for normal dentition. It is a well-recognized fact that,

occasionally, children are born with one or more teeth, and the omens that have been attached to this, or to the appearance of teeth before the fifth or sixth month of life, are numerous. Some claim that it is a favorable sign of longevity, strength, good teeth, etc.; others claim the opposite. These cases occur in the experience of nearly every practitioner, and their importance has been magnified, not so much by physicians as by historians, especially by those who have studied their history from the stand-point of predestination. Premature teeth can be divided into three classes, each of which is due to some alteration in the action of one of the fundamental causes for the eruption of teeth. Changes in the embryonic structures produce teeth without fangs that hang more or less loosely, and are attached by a strip of mucous membrane only. An unnaturally small amount of covering to a tooth will cause it to appear long before its time, and, finally, too great or too early deposit of calcareous material will produce the same result. It occurs, sometimes, that more than twenty primitive teeth are formed, and then one or more may be found prematurely in the mouth. Primitive teeth, produced as the result of the first two causes, are, as far as we know, of no prognostic significance. Those due to premature calcification are said to denote premature ossification of the bones of the head. "As a general rule, however, premature appearance of the teeth is connected with premature ossification of the bony system in general, and of the fontanelles and sutures of the cranium in particular. When this is the case, the upper incisors, as a rule, appear first, undoubtedly in connection with the fact of the premature ossification of the upper part of the cranium. This is a serious occurrence. When premature ossification is congenital, it makes parturition difficult and renders the child idiotic or epileptic." (Jacobi, "The Intestinal Diseases of Infancy and Childhood," pp. 102-103, 1887.) While all this may occur, and undoubtedly has occurred, yet,

according to my own observation, it must be the exception. If any change about the head of a child with premature teeth is to be noticed, it is just as apt to be in the direction of hydrocephalus as the opposite; therefore large fontanels and diastatic sutures. It is now over fifteen years since Jacobi first called my attention to this combination of irregular teething and idiocy. Since that time I have examined into the history of every child whose two upper incisors came through first, and also into the relation of the teeth in idiots with premature synostosis. The latter instances are comparatively rare, but in not a single instance could the mother be positive that the upper teeth appeared first. In several large-headed idiots, or, rather, idiots with widely-open fontanels and diastatic sutures, I have been able to elicit the fact that the upper teeth had appeared first, but not in a sufficient number of cases to establish any law. It has always seemed to me to be purely a coincidence. As I have not kept record of these cases,—it being almost impossible to do so,—my statements, as opposed to those of so reliable an observer as Jacobi, must go on record for what they are worth, to be verified or overthrown by future records.

The question of what is to be done with these premature teeth is one that is deserving of close attention. It will not do to put down the absolute rule that all premature teeth must be pulled. There are positive indications when one of these teeth should be extracted. When the tooth dangles in the mouth attached only by mucous membrane, there can be no hesitation about severing its connection and getting rid of a foreign body that is absolutely useless. When these teeth are tightly set in the mouth, it always becomes a question what to do with them. The rule can be made that they are to be let alone unless some special indication exists for their extraction. I cannot agree with Fleischmann when he says that "the supposition that children who have teeth can hurt the nipples

of their mothers can hardly be taken seriously" (*loc. cit.*, p. 78). There is no doubt that children with teeth do hurt the nipples in more than one way, and that they do not cover their teeth with the lips, as Fleischmann believes. It is true that, in nursing, the two lower teeth are covered by the tongue, but as soon as the upper incisors appear the baby begins to bite at the nipple, and, with women whose nipples are not perfectly normal or thoroughly protected, it is only necessary to see where the fissures are formed in order to convince oneself how these cracks were produced. But, in addition to this, these premature teeth are sometimes situated in such locations as to render all attempts at nursing painful and occasionally futile. When it comes to a question, then, of extracting a tooth or jeopardizing the life of an infant, or even its thriving, the way is perfectly clear, and there can be no possible difference of opinion as to the course to be pursued. The following two reasons are the principal ones for saving these teeth: first, we are never perfectly sure that we are not producing a loss which will not be repaired until the seventh or eighth year of life. The premature tooth that has been extracted may be the only temporary incisor that the child will have, and no tooth will be formed until the permanent incisor makes its appearance; secondly, children with premature teeth may be puny and delicate, perhaps syphilitic; in such children, hemorrhages follow the slightest disturbance of continuity of tissue, and several cases are on record in which children have lost their lives by the extraction of these premature teeth. Magitot, who lost a child after extracting two premature incisor teeth (*Gaz. des Hôpitaux*, 1876), puts down the rule never to extract these teeth, as the result of his unpleasant experience.

Rickets is the most common cause of delayed dentition. But, as has been pointed out before, not every child that gets its teeth late has rickets. The effects of rachitis upon the

teeth are many or none at all, depending upon the amount of rachitic changes that takes place in the bones of the jaw. If, as occurs not infrequently, the bones of the head are not at all attacked by the rachitic process, we can certainly not expect any lesions about the teeth. In such cases, rachitis may develop after the time when the first six or even first twelve teeth have made their appearance, and then there will be, practically, no delayed dentition. An attack of rickets coming on at so late a time may delay the eruption of the following teeth, but, manifestly, this is accomplished by constitutional derangement only, and not by any local effect. But rickets is by no means the only cause that produces late teething. Any disease producing a disturbance followed by diminution of nutritive supply to the teeth will result in a late eruption of the teeth, provided all the other factors for dentition are present in normal quantity. As such we must reckon all disturbances accompanied by long-continued fevers, all long-continued diarrheas, all so-called cachexias. It will frequently be seen that one or more teeth come through during an attack of scarlatina, typhoid fever, etc., and this does not militate against the view just expressed. If this patient be kept under observation for some time, the next group of teeth will be found late in their appearance, and, possibly, deformed.

A more common cause for this late eruption of teeth is to be found in heredity. In another place I have recorded three generations of people; the first of these was decidedly rachitic; square head, bow-legs, etc.; the second, with the same bony malformations to a less degree; and the third, with the characteristic square head without ever having had any symptoms of rachitis. This is, what seemed to me, a well-marked example of the law of heredity. In the same way we can imagine late teething set up in a family, by some affection, perhaps, that has acted through several generations, and resulting, ultimately, in a hereditary tendency. Early or late

teeth are just as distinctive of families as are good or bad ones.

Defective food-supply (*i.e.*, in calcareous material) is one of the conditions accused as the first cause of the establishment of a hereditary tendency. Thus, a deficiency of lime has been held to result in late or bad teeth. This is perfectly correct, theoretically; but, as a result of years of experimenting upon the human being, I have come to the conclusion that we are not in a position to directly affect the teeth by any remedy we may give. Any one of the so-called proximate principles of the tooth may be given indefinitely to a child, and the effect upon the tooth is *nil*. We may (and it has been done by many) produce conditions in the lower animals which will affect the teeth most decidedly, but such artificial conditions can be conceived of as in relation to the human being only in very exceptional cases. It would be difficult to conceive of a condition, for instance, in which all earthy material is absent from the food, unless we would take absolute starvation into consideration. It is highly probable that a deficiency of earthy salts in the food extended over a great period of time does affect the teeth, but hardly in the direction that is under discussion at present. There can be no doubt that children are made rachitic by deficient salt-supply, but this deficiency rarely manifests itself in time to affect the first teeth; in other words, when the food is so bad as to affect the teeth to the extent of preventing the small quantity of calcareous matter needed to cause them to make their appearance in the mouth, the child will not survive. The effects of drinking-water can be taken into consideration only as regards the permanent teeth, since children at the breast do not receive enough water to produce any changes in the teeth. As far as the permanent teeth are concerned, no conclusive relation has, as yet, been established.

There is a good rule for the time of the eruption of the

permanent teeth : they appear in the same order as the milk-teeth and at a number of years corresponding to the months of the milk-teeth, with the exception of four teeth,—the first molars. The latter teeth appear about the sixth year ; they are the first to come through after the second molars in the temporary set, and are found directly behind them. Changes take place in the temporary teeth while this is going on ; the bony septa between them and the permanent teeth are being absorbed, their blood-supply is being cut off because of the obstruction of their arteries, and gradually, as the result of absorption, they are ready to drop out. Momentous alterations in the whole economy are ascribed to these changes by some authors, for which there is even less reason than for those attributed to the temporary teeth, and which must be looked upon in all cases as cerebral,—either upon the part of the physician or upon that of the patient.

As there are thirty-two teeth in the permanent set, the above rule is one that can be accepted only in the rough, but, nevertheless, is one that facilitates memory very much. The second teeth to appear are the canines, from the seventh to the eighth year. These are followed by the bicuspids or premolars, about the tenth year ; then the canines, about the twelfth year ; and finally the molars, from the twelfth to the twenty-fifth year.

A table would be constructed as follows :

First Molars.	Incisors.	Bicuspids.	Canines.	Second Molars.	Third Molars.
6 years.	7-8 years.	9-10 years.	12-14 years.	12-15 years.	17-25 years.

Symptomatology.—The fact that teething is a physiological process has given rise to a peculiar line of argument,—viz., that for this reason no harm can come to the subject as a result of

the appearance of the teeth. On the other hand, the violent adherents of teething sicknesses have overlooked the equally well established fact that morbidity of children is greatest during the first year of life, and so we have developed the two extremes which have been mentioned before. The whole discussion of the symptomatology of dentition becomes a very difficult one because the reasoning *post hoc ergo propter hoc* has been constantly employed, much to the detriment of our conclusions. As far as the physiological nature of the process is concerned, it is but necessary to be reminded of the daily observation that every act which is physiological may become pathological, and, as a matter of fact, has been observed as being pathological. So that from the stand-point of deduction from other processes of the same nature alone we must admit that pathological teething, *dentitio difficilis*, does exist. It is the province of observation to determine to what extent and in what direction these symptoms of a normal process become abnormal are developed. It is of equal importance, in addition, to establish the connection that exists between the process and the symptoms, and, if possible, to establish this connection by facts. Teething, in a healthy child, produces very few symptoms of any kind. This is admitted on all hands for the incisor teeth ; it is claimed, however, that the molars and the canines always produce symptoms more or less intense in their nature. These symptoms can be grouped under two headings for convenience of discussion,—local and general. The local signs are said to be : salivation, redness, pain or itching, swelling, even ulceration. If we examine carefully into each one of these symptoms it may become possible to determine the relation it bears to teething. The history of the physiological flow of saliva in an infant is as follows : The maximum amount—*i.e.*, the greatest quantity—of saliva which flows from the mouth is found between the third and fourth month of life. Before this time it is *nil*, and from this

time it begins to diminish. Now, this physiological flow of saliva is ascribed to an irritation produced upon the lingual branch of the fifth nerve by the lower incisor teeth. As a result of this irritation by teeth that are still within the jaw, but which are supposed to be growing rapidly so as to make their appearance within the next three or four months, reflex action is set up through the glosso-pharyngeal and facial nerves, producing an increased activity of the salivary glands. All this produced by the incisor teeth, which cause so little irritation that the baby is put to bed at night without a tooth and taken up the next morning with one of its little incisors through! So few symptoms present that the watchful nurse, the anxious mother, and, possibly, the doctor have not even surmised the possibility of the child's teething, although they have been on the lookout for three months, since the salivation began! Now we come to another view of the question. The molars are about to come through ; they have broad surfaces, four points, and if any tooth can irritate the mucous membrane of the mouth this is the one that does it. But if there is any salivation, it is very little compared with that of the third or fourth month of life; when it does occur, it is always due to some inflammatory changes in the mucous membrane of the mouth. The salivation, then, cannot be due to teething as a result of reflex mechanism. Its cause is a different one: the salivary glands are developing; any irritation is sufficient to set up a flow of saliva; the cortical salivary centres, the inhibitory centres, are badly developed at this time of life; and, lastly, the child has not yet learned what to do with this fluid which it has not been accustomed to have in its mouth heretofore.

The other local symptoms depend largely upon the nature of the child for their development. It is certain that a great many children get all of their teeth without the development of any local signs whatsoever. The physician must be on his

guard not to accept as signs of teething all the many combined movements of hands and mouth that have been put down in midwifery lore as characteristic for this period. The putting of fingers into the mouth by the child may mean very much or very little. It may mean that the child has learned to use its fingers for the gratification of its highest pleasure and aim in life,—sucking; it may mean that there is some irritation, better pruritus, about the gums, for when there is pain, as in stomatitis ulcerosa, the child is very careful not to put its fingers into its mouth; finally, it may mean irritation very much deeper than the gums.

The fact that children do have pains during teething cannot be denied, but the case of the eruption of a wisdom-tooth in an adult is not to be looked upon as going very far to prove that a child ought to have pain when it cuts a molar or an incisor. The wisdom-tooth in an adult is, to begin with, a more or less rudimentary organ, badly developed, frequently diseased, and in the lower jaw crowded through space not sufficiently liberal, bounded in front by the second molar tooth and behind by the lower jaw. Would a comparison with the pain produced by the permanent teeth not be a much fairer estimate of the number of children that suffer from teething? Observations in this direction would show that pain, swelling, redness is by no means as common as is generally supposed.

The remote symptoms that have been ascribed to teething are many, and in this connection are found the many impossible and improbable combinations that have played such sad havoc in pediatrics. In the present state of our knowledge it cannot be expected that a great many of these be discussed seriously, nor will it be at all satisfactory to any reasoning person to claim that because a certain disease occurs while a child is teething, this disease must necessarily be due to teething. The etiology of disease is by no means a closed book, but enough has been done to exclude impossible combinations.

Any one claiming that an inflammation of a remote organ is due to teething, for example, would have to be able to make out a very much stronger case than would be implied in the mere statement of the two facts,—teething and inflammation. A great many of these combinations still exist, and they exist principally because authors deem it their duty to copy what has been written by others without sifting the evidence and without bringing their own experience to bear upon the subject. Again, it is difficult to rid one's self of preconceived ideas, and one who has been taught that certain combinations exist will find it a great task to discover the existence of something beyond the two combined points. On the other hand, one who has not been taught these combinations or who has disregarded them will, possibly, be just as likely to err in the opposite direction,—to disregard them. For these reasons an impartial judgment is very difficult.

What was said of the local symptoms may be said with equal propriety of the general symptoms: they depend for their development upon the nature of the child. Given a poorly-nourished, badly-developed child, or one with a distinctly nervous, hereditary tendency, and it will suffer very much more from anything than one perfectly healthy. A child of this description will be less able to endure pain, will suffer more, and will, therefore, react more decidedly than one better prepared by reason of good condition.

A teething child, when it suffers at all, will be found to have changed its disposition for the time being. It becomes irritable, fretful, cross, difficult to amuse, has less appetite than usual, its sleep is more or less disturbed, and presents all the signs of what might be termed malaise in a young child. With this there may be a slight elevation of temperature, increased thirst, and rapidity of pulse. This condition usually precedes the eruption of a tooth and disappears suddenly either before or after the tooth has come through. The more

irritable the child is, naturally, the more marked are these symptoms.

The symptoms on the part of remote organs can be grouped under the following heading: Symptoms on the part of the nervous system, the digestive apparatus, the skin, the respiratory apparatus, the genito-urinary system, and the organs of special sense. For the nervous system it is principally convulsive disorders, partial or complete, that are ascribed to teething. The physiological facts that are brought in to explain these convulsions are as follows: In young children the inhibitory power of the brain is very much less than in adults, therefore any afferent impulse would be followed by a very much greater reaction than in the adult. This reaction is supposed to manifest itself in the form of generalization of reflexes,—*i.e.*, convulsive movements. While these facts are perfectly correct from a theoretical stand-point, it does not follow that their application is absolutely so. In the case of teeth the afferent impulse is carried through the fifth pair of nerves to the medulla, and from here it is carried as efferent impulse by the nerves of the face and also the spinal nerves, not being restrained by the normal inhibitory power. The force that generates the nervous impulse is supposed to be pressure upon the peripheral termination of the nerve in the mouth. In order to get a convulsion from teeth, two things must be taken into consideration: First, the amount of pressure or irritation; secondly, the irritability of the nervous system, the whole or various parts of the reflex arc. It seems to me that the first element can be excluded, in that it alone could not quantitatively be held accountable for the convulsion. Such a statement is only justifiable as a result of comparison with something that is tangible, with some disease in which we are qualified to judge of the quantity of pain there is present. It is next to impossible to judge of how much or how little pain is produced by the tooth's pushing through the mucous

membrane; the probabilities from a theoretical stand-point being that this quantity is very small. The reason for this statement will be found in the fact that the tooth has been pressing upon the nerve filaments for some time, producing functional paralysis, if not atrophy. It is more than probable that the pain of teething is the result of the catarrhal stomatitis that is always present, or of pressure upon neighboring teeth or tissues. In phlyctenular conjunctivitis or keratitis we have a disease in which we know that the pain and irritation are very great, and yet no author claims that this disease is followed or accompanied by general convulsions. We do find a tonic contraction of the orbicularis palpebrarum, but nothing more. It seems to be stretching a point to make the irritation of a tooth produce general convulsions, and the greatest irritation of one of the most sensitive organs of the body to be followed only by a local disturbance. Teething convulsions can be produced only, then, by a too great irritability of the nervous centres; unfortunately, this can neither be proven nor disproven, so that there is nothing left but to take the statement of authors and to have recourse to observation. The authorities vary very much, and while, theoretically, the possibility of the production of general convulsions from teething cannot be denied, the probability of such a combination must be rejected. For myself, I am free to confess that I have never seen a case of teething spasms. In every case of convulsions that has come under my observation it has been easy to detect a much more plausible cause, which, when removed, caused the convulsions to cease. If we resort to the following method of determining the cause, however, our conclusions are not apt to be very convincing. Say we rub a tooth through in a patient ten months old; patient being fed upon improper food,—slightly constipated; after having given a dose of calomel and the convulsions have stopped, it is not fair to ascribe the spasm to the poor, maltreated tooth.

Yet this is being done daily, and the physician is not willing to admit that the convulsion is most likely caused by the absorption of some substance from the intestine, and not by the tooth. Even if the tooth fails to make progress and the wound which has been produced by the rubbing heals up, so that the local conditions are the same as they were before the doctor interfered,—and the child ought to have convulsions all the time,—the course of reasoning is not altered ; a child with convulsions, a doctor to rub, convulsions to cease, therefore the rubbing doctor cured the convulsions due to a tooth. For practical purposes it is much safer to say that teething never produces convulsions than to hold that convulsions, as a rule, are caused by teething. Even the manifestation of local convulsive movements ought to be carefully examined into before a positive conclusion is arrived at. It is not going too far to state that, as a cause for convulsions, teething ought to be looked upon as the last etiological factor and not as the first. Only after every other possible source has been examined into ought we to be willing to admit teething as a cause for spasms, since it is necessary to conceive of something,—a something that may exist and possibly does exist, but a something which is very rare and, at the present, thoroughly unknown.

On the part of the digestive apparatus all forms of trouble have been attributed to teething, from the ordinary dyspeptic vomiting to a general affection of the whole alimentary tract. The mechanism of these lesions has been principally attributed to the irritation that follows the swallowing of great quantities of saliva. From what has been said before, it will be seen that this cannot by any possibility be the true cause, for the flow of saliva is usually very much diminished or has entirely ceased by the time that teeth make their appearance. In connection with diseases of the alimentary tract the teething theory has been followed by the most pernicious results. It would not be in accordance with daily observation to say that

teething does not have any influence upon the bowels, but this is to be by no means understood as intimating that it is frequently or directly the cause. The only effect upon the bowels is that they participate in the general irritability of the child. Just as the skin may become hyperæsthetic, so the bowels may become less tolerant, and an absolute adherence to physiological food will soon clear up the bowel complaint. In other words, at this time, the digestion will be interfered with to such a limited extent as to cause food not strictly proper, but well borne at other times, to produce disagreeable effects upon the bowels. It has been pointed out that teething diarrhoea has peculiar properties, so that it can be differentiated from other forms. I must confess that I have failed to find these. The stools may indicate a disturbance in any one or more sections of the bowel so as to be characteristic or not; the disturbance is of very short duration and always amenable to a strict diet. In some children constipation is supposed to be produced by teething; this, however, is most probably a coincidence. There is one thing that ought not to be forgotten in connection with the intestinal lesions produced by teething: the children are old enough to come to the table, they make known their wants, sometimes are able to satisfy themselves by getting what they want, and in every respect are most unlikely to be kept upon physiological food. There is no question of the fact that the more carefully children are watched, the more carefully they are fed, the less liable are they to teething diarrhoeas. It has always been my firm conviction that if a child were kept upon absolutely physiological food, that child, provided it were otherwise healthy, would not have diarrhoea from teething. Here, as before, it cannot be too strongly insisted upon that the physician look to other causes besides teeth before he takes it for granted that he is dealing with teething diarrhoea. The peculiar fatalism of allowing a diarrhoea to go on because it is due to teething

is perfectly unintelligible. The "checking the bowels" can do no harm, and danger can and does arise when the catarrh of the intestines is allowed to continue. Many a case of tuberculosis can be traced to this pernicious doctrine, many invalided children owe their bad health to this preconceived notion, and many a life could be saved if only the trouble were taken to institute proper feeding, which can certainly not be harmful; this method can be pursued even where the physician objects to giving medicines to stop the diarrhoea. In addition, the doctrine engenders carelessness, both with physicians and laymen, so that grave lesions are frequently overlooked.

The symptoms on the part of other organs must be ascribed to coincidences. It would take a vivid imagination, in the present state of our knowledge of inflammation, to conceive of a production of a bronchitis by the wetting of a child's breast from the saliva, which is supposed to be present during teething. The same may be said of the presence of the gonococcus in urethral or vaginal discharges, which are supposed to be due to teething. As for the skin, young children do have delicate skins, the least irritation may produce a general eruption. A flea-bite will cause the child's skin to be covered with an erythema or an urticaria; the same can be said for a bed-bug or any insect sting. It is logical to conceive that an eczema may be set up by the irritation which follows a constant outpouring of saliva over the skin; but we fail to find any connection between teething and the various other forms of skin trouble that have been attributed to it, such as lichen, herpes, pemphigus, etc. The mistakes that are made in this direction frequently become ludicrous. It is not long ago that a child was brought to me with a "teething impetigo" which, notwithstanding the teeth had appeared and notwithstanding the internal use of all sorts of remedies, would not get well. To the great astonishment of all concerned, a needle put into

one of the pustules succeeded in bringing out a small black body, which, under a magnifying glass, disclosed itself as an acarus scabiei.

In teaching I have, for years, put down for my students an axiom that "teething produces teeth and nothing more." While theoretical conceptions cause us to deviate slightly from this position, it will be found that the more acute the diagnostician, the more accurate and searching the examination, the nearer the truth this statement.

Treatment.—It would be waste of time to discuss the various means and remedies that have been proposed in order to make teething easier. Whether we give to the child a hard or a soft body to put into its mouth is a matter of the utmost indifference, but one thing ought to be insisted upon: nothing which has a bad effect upon the digestive organs should be given to the child to facilitate teething. The only remedy at our command to insure normal teething is to keep the child in good health. To this end, at the time of teething as well as at all other times, the child must receive proper food, sufficient oxygen, be kept clean, and clothed properly. Such a child, without hereditary tendencies or acquired disease that may affect the teeth, will not be disturbed to any appreciable extent by teething. If we find that the child becomes irritable and cross at the time when teeth can be reasonably expected, it will be found advantageous to redouble our attention as to diet. At this time also, instead of keeping the patient in-doors for fear of catching cold, the opposite policy will be found very serviceable. Send the child out into the air as much as possible; nothing acts so well in a fretful child. It is not necessary to bundle the child up to suffocation. The dress of the patient should be suited to the season of the year; no bandages, no flannels, no woollens are necessary in summer; the cooler the child can be kept the more comfortable will it be. In winter the child should be dressed warmly; it is not

necessary to protect any especial organ against any especial disease, either at the teething time or at any other. A great deal of discomfort could be spared children if laymen as well as physicians were able to shake off some of the old superstitions connected with the theory of catching cold.

If the child should have increased temperature, the luke-warm bath will not only control it, as a rule, but will also assist in removing the cause of the fever. In order to remove the cause effectually it will be found necessary, in some cases, to administer a laxative,—rhubarb or calomel,—to be followed up by washing out the large intestine. In other cases, where there seems to be no trouble from bad feeding (and this can be determined only by the strictest search into the history, including, if possible, an examination of the stool), it will be found that the internal administration of the bromides will give the patient much relief in that it seems that the fever is due directly to some effect upon the heat-centres. Every one who has examined into the subject of infant-feeding will, I think, admit that no positive conclusion can be arrived at regarding the food of any ordinarily kept child without examining the stools. If those who are so quick at drawing conclusions between teething and diarrhoea would take the trouble to search the stools carefully, they might find reason to change their opinion. It is a very common occurrence to have both the mother and the nurse disclaim any error of diet, and to find, upon examination, a piece of undigested potato, a bit of apple, or some other equally unphysiological body in the faeces. Above all, it is wise for the physician to wait a little while before he makes up his mind that he is dealing with a dentition disease, so that he may examine his case carefully before he comes to a conclusion which has few chances in its favor. If this conclusion should be arrived at as a *dernier ressort*, the diarrhoea must be treated as any other diarrhoea would be. Whatever sovereign remedy the physician has he

must use, especially in hot weather. Without wishing to disparage the use of the so-called intestinal antiseptics, I am free to confess that, to my mind, the profession is going too far in giving up the use of opiates in intestinal disorders of children. After the cause of the diarrhoea has been removed mechanically by washing out the stomach or the intestines, there is left in the stomach or intestines an anatomical change which opium, in its action, is especially fitted to benefit, and which is very little, if at all, affected by antiseptics. Without going too far in this discussion, it is but proper to state that we are not warranted in giving up the results of years of experience for a theory not, as yet, properly proven nor thoroughly worked out.

No one remedy has been considered so much in the light of a specific against the maladies of teething as lancing the gums. More especially is this true among English-speaking people. Emanating, as we will see, from a celebrated French surgeon, it was taken up and diffused by the English, and in this way has come to us as an inheritance of more than doubtful value. While with us the scarification of the gums is by no means as commonly done to-day as formerly, yet there are very many excellent practitioners who still resort to this remedy both as a matter of routine and from conviction of its utility. The names connected with the historical development of the subject of lancing the gums are principally three in number,— Ambroise Paré, John Hunter, and Marshall Hall. The first named has been neglected by most writers upon the subject (notable exceptions being Fleischman and Finlayson, although the latter's description is not correct), the second is universally referred to, and the third seems to get more credit for his amount of work than is due. The first mention of gum-lancing is found in the edition of 1579 of the works of Ambroise Paré, at the end of his book, "*De la Génération*" (numbered variously), and in all subsequent editions and

translations. The statements made are the same as to facts, the language is changed in some of the subsequent editions (French edition of 1585, Guillemeau's Latin edition of 1582), but only as to minor details. Paré mentions the following remedies: "Rubbing the gums with oil of sweet almonds, fresh butter, honey, and sugar, or mucilage, from the seeds of puceron, marshmallow, quince, and on the outside a poultice of barley flour, milk, rose oil, and the yelks of eggs is to be applied: it is of advantage to rub the gums with the brains of a roasted or boiled hare, because experience demonstrates that the gums relax, and, owing to some occult properties, the teeth are helped in coming through; the brain of a dog is also good." Sometimes these and other remedies that he mentions are of no avail, "because the gum is too hard, which is the reason that the teeth cannot pierce them, from which follows, on account of the tension, that the children have great pains, from which follows fever and other complications mentioned above, even death. And therefore I am of the opinion that the surgeon should make an incision into the gum upon the tooth in order that the way is opened for it so that it can come out more easily. This is what I have done to my children in the presence of M. le Féuré, physician-in-ordinary to the king, and of Madame la Princesse de la Roche-sur-Yon, and of Messieurs Hautin and Courtin, doctors regent of the faculty of medicine at Paris, and of Jacques Guillemeau, surgeon-in-ordinary to the king, and sworn at Paris." He then states that some nurses, as is frequently done nowadays, scratch through the gum with their nails "in order to make way for the teeth that want to come through." Paré ends the chapter on generation by reciting the following history which taught him to lance gums. He was called to make a post-mortem upon the eight-months-old child of Monseigneur de Nevers; "having diligently searched for the cause of his death, nothing was found unless it might have been the hardened,

enlarged, and swollen gums." When the gums were cut the teeth were found "ready to come through," and the conclusion was arrived at, by himself as well as by other physicians present, that "the sole cause of death was that nature was not strong enough to pierce the gums and push the teeth out."

Notwithstanding such high authority, the operation has never gained a great foothold in France, and the present status of the question can be shown by a quotation from Barthez and Rilliet: "Without denying the favorable results obtained by other practitioners, we must say that our personal opinion is not favorable to this method of treatment. We have frequently practised this small operation, but we cannot recall a single instance in which it seemed to have any real utility. We will add, in order to be consistent with truth, that to us it has never seemed sensible."

We must turn to England for the full development of the practice of lancing the gums, and we begin with John Hunter (*loc. cit.* p. 609), who was the first to call attention to the reflex irritation produced by these foreign bodies, and, logical as he was, to carry out his treatment to the extreme, lancing freely and frequently.

To Marshall Hall (London *Lancet*, 1814, vol. i. p. 244) is usually attributed the credit of having brought the method into general use. While there can be no doubt of his having fortified the position, yet, from what is said by John Hunter, we are led to infer that in his day the practice was becoming pretty generally accepted. Marshall Hall did the operation because he wanted to relieve the "nervous action" by getting at the nerves themselves; therefore, "it is to the base of the gums, not to their apex merely, that the scarification should be applied. The most marked ease in which I have observed the instant good effect of scarification was one in which *all the teeth had pierced the gums!*" The gums should be lanced once

or twice daily, if necessary ; " better do this one hundred times unnecessarily than have one single fit from the neglect of so trifling an operation." From this, as a starting-point, it will be seen how the process of gum-lancing has entered into the profession as simply a routine method of treatment, the indication for its use being about as follows : All the diseases of infants are due to teething ; all the bad effects of teething are removed by cutting the gums ; therefore all children ill at the time of teething ought to have their gums lanced. It is very difficult to give any adequate conception of the amount of gum-cutting that is still done in Great Britain. In a discussion held in the Medical Society of London, fifteen members took part, of whom nine were decidedly in favor of lancing, three were opposed, and three non-committal. Nearly all of those in favor, however, had receded from the extreme views of Marshall Hall, and had, when they spoke of the subject, put for themselves certain indications, as, for instance, Mr. Hamilton Cartwright, who, although he thinks that convulsions and diarrhoea are due to teething, cuts the gum only when the gum is tense and glistening and the tooth about to come forward, and in inflammatory conditions of the gum, with tumefaction (*British Medical Journal*, November 8, 1884). This discussion gave rise to correspondence, in the same year of the journal, in which letters are printed from quite a number of practitioners in England, the result of which certainly seems to be that the operation under discussion is still used as a routine method by a very great, if not the greater, number of physicians in England. Certain it is that all the modern English books (West, Money, Semple, Day, Ashby and Wright) recommend lancing in a more or less limited way.

The operation has never extended into the medical acquisitions of the laity in Germany. The modern authors of Germany speak of gum-lancing as useless and, possibly, harmful,

and the physicians do not carry gum-lancets in their pocket-cases. In our own country cutting of gums still has its disciples, and especially among dentists, although it is far from being an uncommon practice with physicians. Jacobi (*loc. cit.*) says that "the local treatment of swollen gums, which consists of lancing, has fortunately become less common and popular than it was in former times." It may be taken for granted, however, I think, that the greater number of physicians no longer resort to the lancet as routine treatment. It may, furthermore, be stated that most of our authors advise lancing of the gums in exceptional cases only. One of the most recent and most enthusiastic advocates of the operation is Starr ("Diseases of the Digestive Organs," p. 102, 1886). "If there be fever, nervous irritation, sleeplessness, vomiting, or diarrhoea during the progress of, and dependent upon, dentition, I invariably lance the gum,—provided the position of the tooth can be established by the touch,—making the incision superficial or deep according to the distance of the tooth from the surface." This quotation presents the status of the question, as far as some practitioners are concerned, in a complete way. If certain things are present which, it is taken for granted, depend upon teething, then the gums must be lanced,—a position almost as broad as that of Marshall Hall, who, however, had no ifs, and who was willing to say that these certain things were always dependent upon dentition. This position, however, is the exceptional one among American writers; in contrast with it see Dorning ("Keating's Cyclopaedia"), who says that the good done by the operation in every instance, most likely, "was a pure coincidence or the result of the hemorrhage (a blood-letting) or of imagination on the part of those interested."

For the operation an instrumentarium quite formidable in size has been recommended,—a roughened coin, the lancet, the scalpel, and various instruments especially devised for the

purpose. It will not be saying too much that, when the gums are to be cut, they should be operated upon *lege artis*, with all aseptic precautions. The methods that have been employed, again, are many. Some prefer superficial, some deep incisions ; some cut at the top, others cut at the base of the tooth. The forms of the incisions are principally three in number,—the linear, the crucial, and the elliptical. To these may be added the elliptic incision with a dissecting off of the gum, the cutting across the margin of the gum, and, finally, Marshall Hall's method, which consists in cutting the tissues as deeply as possible.

In the discussion of the subject the following questions can be asked : Does the operation do good, and how ? Does the operation do harm, and how ? In answering the first question we can subdivide as follows : The effect upon symptoms ; the effect upon the process of teething when the operation is performed. There are three classes of authors,—one which believes in scarification unconditionally ; one which does not believe in it at all ; and, lastly, one which thinks it does good to allay some one symptom. Of the latter class, the symptom which is most commonly picked out as being relieved by gum-lancing is a convulsive seizure. It is claimed that gum-lancing cures convulsions. While we are not prepared to admit that convulsions are produced by teething, upon theoretical grounds we have been forced to admit that such a connection might be a possibility. How, then, can a convulsion which, upon theoretical grounds only, is produced by teething be relieved by gum-lancing ? A relief given to the centres cannot be excluded, provided the hemorrhage be sufficiently great. The same can be said for the local process with the same proviso. Blood-letting does relieve arterial pressure ; convulsions produced by brain hyperæmia can be relieved by one or two leeches behind the ear. Indeed, the older physicians were in the habit of differentiating between convulsions due to menin-

gitis and convulsions due to other causes by means of blood-letting. If the convulsions ceased, it was a bad omen ; if they continued, the child might recover. Again, blood-letting empties ptomaines from the general circulation ; if the convulsions are toxic, why should it not cure if the poison in the blood is reduced in quantity or taken out altogether ? But no one would think of choosing the mouth as the place for a sanguinary depletion ; so that this is not the reason ascribed for the beneficial effects of our operation. Locally the scari-fication does nothing except let blood. The idea that tension within the sac is relieved is a purely imaginative one. There is no tension within the sac, and, as has been shown, there is more than enough room for the tooth ; so that under normal circumstances pressure within the sac or upon its bony surrouindings, in all directions, becomes an anatomical impossibility. Under abnormal conditions lancing of the gums has no effect upon the condition within the tooth-sac.

In more or less general terms it has been stated that irritation of the nerves is produced by the tooth.

The mucous membrane above the tooth has its nerves atrophied as the result of constant pressure ; in other directions no nerves are pressed upon ; how, then, can gum-lancing relieve a thing which does not exist ? But most excellent observers state that convulsions are relieved by gum-lancing ; observers whose word cannot be doubted. What has happened in these cases ? Either the hemorrhage has been sufficient, or the reasoning has been of the nature first *post hoc ergo propter hoc*, or the convulsions would have stopped without any interference. As a matter of fact, gum-lancing neither prevents nor causes convulsions due to teething in all instances (see an excellent paper by Cairns, *Edinburgh Medical Journal*, 1869) ; in other words, when the conditions are not propitious the remedy has no effect. It is extremely easy to delude one's self concerning the efficacy of a remedy, especially when one is prejudiced in

its favor. Convulsions in young children are a very uncertain quantity ; depending, as they do, more upon central or systemic than upon local causes, they are apt to begin or cease upon very slight provocations. Who has not seen a convulsion cease as quickly as it came ? It is within the experience of every one to have noticed the application of most simple means apparently cure convulsions. On the other hand, convulsions will be met with that cease only after extreme measures have been used, or, depending upon the cause, do not cease at all. It is safe to say that a great many convulsions cease after gum-lancing that would have ceased without any interference whatsoever. But the advocates of the lancet are not satisfied with this ; "the operation worked like a charm," "the effects were miraculous," and a great many more expressions of like nature are to be found in their writings. A great many forget to state that some remedy had been given before the gum was lanced, which had possibly removed the cause of the convulsion. A great many are so preoccupied with the good results that must follow that their impressions are obscured, and what would be a sudden stoppage of the convulsions would to others be the natural course,—a gradual lessening until complete cessation has taken place. In my whole experience I have lanced the gums once ; then at the earnest solicitation of a consultant. The result was *nil*, and in all other cases that I have seen in the practice of other practitioners the result was the same. Convulsions that can be cured by gum-lancing can be cured by the most simple means,—a lukewarm bath, a mild laxative, a full dose of bromide of potassium ; but the principal factor must not be lost sight of,—viz., that they will get well of themselves if the physician will be wise enough to remove the cause, which, as has been stated before, must be looked for everywhere else than in the teeth. If he wish to do blood-letting, let him get a few leeches or the artificial leech, but let him not ascribe the benefits of a hemorrhage to gum-lancing.

In this, it is not proposed to recommend blood-letting ; this subject is foreign to the one under discussion.

As far as the effect upon a diarrhoea is concerned, it would be impossible to trace any connection between the gum-lancet and increased peristalsis. Here the relief that is given can be explained purely upon false reasoning. We would defy any practitioner to successfully treat an attack of cholera infantum or catarrh of the small intestine by neglecting the laws of diet and relying upon gum-lancing alone. In reading through the accounts given in relieving diarrhoea by scarification, it is always a little gray powder, a small dose of calomel, possibly bismuth, careful diet, plus the lancet. We never find the lancet used alone as the great specific ; so that all deductions drawn in this way must be fallacious from a scientific stand-point. When the physician lances the gums, he immediately gives directions as to the other treatment ; the child is now for the first time treated as being ill, by its surroundings, and the change of diet, the additional care, and the medicines produce the effects that are ascribed to the lancet. As long as a child is simply teething it is good to keep the bowels open, say the old women ; the more you keep the bowels open the better ; therefore the diarrhoea is salutary. Very few people take their children to the physician with diarrhoea, if the diarrhoea happen to occur about the time teeth ought to appear. It is only after they see that the child is suffering in appearance and general health that they find it necessary to consult the physician, —frequently too late for him to do anything ; but after he has lanced the gums they go away perfectly contented, although the child does not improve, and, possibly, may lose its life. If the physician, on the other hand, gives the proper directions, with or without the lancet, the child is apt to recover. Certainly, in my own experience, with others, the lancet has never stopped a diarrhoea, and, I am equally certain that, with the proper remedial agents, without the lancet, failure has been comparatively rare.

That the operation makes teething easier or more rapid is a point that has been frequently urged. That teething is not facilitated is shown by all those statements that are made in regard to the number of times it is necessary to scarify. It is a very difficult matter to state which tooth is coming through, and more difficult to estimate the time when it will come through. More than once have I seen the wrong tooth lanced, and frequently teeth lanced that did not come through for months afterwards. How the eruption of teeth is made more rapid by scarification is difficult to conceive. The movement of the tooth is from below upward, or from above downward, as the tooth happens to be in the lower or upper jaw. If, now, we cut the gum we do not in any way facilitate this motion, for the gum does not offer any resistance when the tooth is ready to push through it; certainly none that the moving tooth does not easily overcome, and the motion itself can certainly not be increased by the gum-lancing. We are practically digging a hole, expecting to remove the object at its bottom without raising it.

We have seen that lancing, *per se*, does no good. Does it do harm? The operation itself may be harmful in that, first, it produces a wound where there should be none; and, secondly, by hemorrhage. In these days of antisepsis we are loath to make wounds when they are not necessary, and, I dare say, a great many cases could be found in which infection of the scarified gum has taken place, producing much more damage than would have been done by the teeth. In an article by Behrend (*Journal f. Kinderkrankheiten*, iii. 6, 1844) we find a case of this sort mentioned which occurred under the eyes of Marshall Hall. Behrend, who was in England at the time, makes a strong point of the unnecessary risk that children are exposed to from this cause. The cicatrix that is produced as the result of frequent gum-lancing has been spoken of as being another reason why the operation should not be performed. It

does not seem, however, that a cicatrix produced in this way should offer very much resistance to the pushing tooth. On account of the comparative freshness and low vitality of this tissue the resistance might even be looked upon as less than that of the normal gum, although the latter is small enough.

The danger from hemorrhage has certainly been underestimated. Although the number of directly fatal cases is sufficiently great to warrant care in this direction, it is not only directly, but indirectly, that hemorrhage kills, as children will bear loss of blood much less than adults. In looking through the literature I have found ten fatal cases of hemorrhage, besides two others (Hamilton and B. W. Richardson) mentioned by James Finlayson (*Brit. Med. Journal*, September 19, 1874). Of these ten, five have been mentioned before by Finlayson,—they are those of Taynton, Anderson, Whitworth, Des Forges, and Nicol. The new ones that are added are those of Bonney (*Lancet*, 1854), A. C. Castle (*Boston Med. and Surg. Journal*, 1849), two cases of J. W. Garland (*ibid.*, 1878), and one case of Yale (*ibid.*, 1878). So that twelve cases have been recorded in which death was due directly to this operation. That there are a great many more that have not been recorded no one can doubt, for in all these years it is certain that more than twelve bleeders have been lanced, in each of which the hemorrhage would probably have been fatal. The greatest number of cases in which excessive hemorrhage does harm are not reported at all; we refer to those that produce acute anaemia (Behrend, Churchill, Barthez and Rilliet, Finlayson). These cannot be represented in tables or by statistics,—anaemia which produces other changes, which affects the digestion, the whole metabolism of the child, which causes the child to be less resistant to external noxious agents. All this should not be underestimated; and when authors speak of the harmless incision of the gums, an operation of no moment, we are willing to agree with them as to the great majority of cases, but

must insist on the direct harm that is done in a much greater number than is usually admitted. From our stand-point gum-lancing does good only as a local or general depletion, and as such it ought never to be used. Finally, there is another aspect of the question which has already been referred to. It is the harm that is done by preventing good. It is the error that is constantly inculcated. It is the making of a routine practice, based upon purely theoretical assumption, which is in the way of careful diagnosis and individual development. A man who has settled himself to the belief that teething produces all the ills of childhood rarely gets beyond this ; if he, logically, lance gums, he can see nothing more in the therapy of nearly all diseases beyond this. Cairns (*loc. cit.*) says that gum-lancing "tends to perpetuate a custom which, to say the least of it, is of a doubtful character ;" I would add, which is useless. The conclusions that we would arrive at in regard to gum-lancing are as follows :

- I. It is useless, *a*, as far as giving relief to symptoms ; *b*, as far as facilitating or hastening teething.
- II. It is useful only as bloodletting, and ought not to be used as such.
- III. It is harmful, *a*, in producing local trouble ; *b*, in producing general disturbance on account of hemorrhage ; *c*, in having established a method which is too general to do specific good, and too specific for universal use.
- IV. It is to be used only as a surgical procedure to give relief to surgical accidents.

IX.

THE TONGUE AND THE MOUTH IN DISEASE OF
REMOTE PARTS.

THE mouth can be so easily examined that it affords certain guides, more or less universally accepted, to diagnosis. The older physicians were very careful about the examination of the mouth, especially the tongue; but at present, the diagnostic value of certain changes is largely disputed. While it would be difficult, in individual cases, to base a diagnosis upon the appearance of the mouth, yet there are to be found combinations of appearances which leave little or no doubt as to the disease which produces them. We are disposed to smile incredulously at some of the descriptions found in the works of fifty years ago, yet the methods of exact diagnosis have multiplied so rapidly, and purely clinical evidences are so often neglected, that it is questionable whether we have a right to do this in all instances. Certain it is that the older physicians, with their limited means, made diagnoses that were very wonderful; and equally certain is it that we, in our generation, with all our physico- and chemico-medical means, overlook very important conditions. Indeed, it might be said of us that, on account of all these diversions, purely objective examination is on the decline; whether a forward or retrograde movement it is not our purpose to discuss, but where the older physician would examine the tongue in a case of typhoid fever, the modern examines for the bacilli in the dejections. We have gone through the periods of pure clinical medicine, the medicine of pathological anatomy, and are now in the throes of etiological medicine. Every time we go through one of these periods, each one making a decided advance, a little is dropped of that which has been common knowledge, principally because

this common knowledge cannot be made to agree with the theoretical views held at the time. Each new method of examination supersedes some old one, possibly not covering all the ground of the older one, but, nevertheless, that knowledge gained by the older one, and not gained by the newer, is lost sight of.

One of the things that has been treated of in a step-motherly way is the examination of the mouth. It is true that no patient considers himself thoroughly examined until he has "stuck out his tongue" at the doctor ; but usually the examination is performed in a perfunctory manner, and the physician gains very little knowledge. On the other hand, there is that class of physicians which makes the examination of the tongue the principal basis for diagnosis, and in intestinal troubles this and an examination of the stools seem to be sufficient to give a clear insight into the case. It seems hardly necessary to state that the truth lies in the middle ; that in some cases the tongue is of great clinical importance, and that in a great many others its examination for diagnostic purposes is without value. It goes without saying that no case is completely examined unless the mouth has been looked into, and yet, in a great many cases, nothing is gained by this examination.

In infants the tongue can only be examined by looking into the mouth, and the same rules that have been put down elsewhere hold good for this examination. The appearance of a normal infant's tongue has also been described in a previous chapter, so that at present we are engaged upon a discussion of the tongue in disease. The tongue is affected as the result of local or general conditions. The changes that take place are in the direction of size, shape, color, and coating or fur. As the tongue is a muscular organ, endowed with both nerves of special sense and nerves of motion and sensation, we may have changes which affect either one or all of these structures, producing paresis or paralysis, loss of taste or sensation. Loss

of motion is easily diagnosticated in children ; not so with loss of taste or sensation ; in infants the latter would be almost impossible, in older children not so difficult.

The tongue changes its size and shape principally as the result of the action of local causes. It becomes too large in glossitis ; it is somewhat swollen in those forms of stomatitis (catarrhalis, ulcerosa) in which its mucous membrane becomes infected, and this infection is carried into the body of the tongue. Glossitis is an extremely rare affection in children, due, possibly, to the absence of causes acting principally during adult life. Congenital largeness of the tongue is not rare ; this is usually associated with one or the other form of idiocy, and the open mouth, with the large protruding tongue, the saliva running out of the mouth, is sometimes sufficiently characteristic to lead in the right diagnostic direction. Abnormally small tongues are usually the result of malformation, and are very rare.

The size of the tongue usually affects its shape ; it is an innate tendency to keep the tongue within the mouth, and it is only under abnormal conditions that it is found protruding for any great length of time. Being confined, the teeth leave their impression upon the tongue's border, and, furthermore, as long as the tongue can be retained within the mouth it is usually much swollen in its vertical diameter. These conditions are somewhat different in children, but it is not uncommon to find the marks of the teeth upon the sides of the tongue.

The blood affects the color of the tongue, as a whole, more than any other cause. When the blood cannot be returned to the general circulation from the tongue, this organ becomes cyanotic, of a slight but decidedly bluish tint, or even purple. Constant and persistent coughing (pertussis) produces this effect, and the color of the tongue is sometimes of great value in establishing this diagnosis. In measles this change

has already been referred to before, although it seems to have been overlooked by other authors. Monti (*Jahrbuch f. Kinderheilkunde*, N. F., vi. p. 27) says, "The tongue does not participate in the diseased process of measles." Whether this slight cyanosis is due to the cough that always accompanies measles, or whether it is due to some change within the tongue itself, I am not prepared to state.* It is present in all the cases of measles that have come under my observation for some time; but it seems impossible to disassociate it from the act of coughing. So much, however, may be added, that in cases of bronchitis, in which the cough seems to be very much more violent than in many cases of measles, the bluish discoloration may be absent. As a symptom of general cyanosis, a blue tongue is of some importance. Reference need only be made to the diagnosis of skin discolorations in the colored race, and this statement becomes very apparent when the statement is made that I know of no way by which the diagnosis of cyanosis can be so easily made in a full-blooded negro child than by examining its mucous membrane. Even in white children the cyanosis of heart trouble or pulmonary affection, especially the chronic forms, is seen to great advantage in the mouth.

The absence of color, or paleness, is caused by all those conditions which produce anæmia. As a result of hemorrhage, the tongue may suddenly become comparatively colorless. In Hodgkin's disease, leucocythaemia, chronic anæmia, the cachexia of malaria, the tongue is markedly pale. In all wasting diseases of children the tongue seems smaller but decidedly changed in color. It is, however, the chronic forms of disease especially that produce this change in the color of the tongue as a whole; acute processes either do not produce it or it is

* It seems to me, after repeated observations, that the eruption of measles appears upon the tongue, as it does upon other parts of the mucous membrane of the mouth.

masked by the coloring given to the mucous membrane. It is very difficult at times to get an accurate idea of the color of the tongue-substance; this, naturally, being more or less changed by conditions of the mucous membrane. That part of the tongue resting upon the floor of the mouth is, manifestly, more available for this purpose than the dorsum; in very young children it is difficult to get at, and in older ones, where there is inflammation in the oral cavity, the filling of the blood-vessels masks the color of the tongue.

The furring of the tongue is that portion of our subject that has been most studied. The fur upon the tongue is, when examined microscopically, seen to be made up of epithelial cells, molecular detritus, and organisms of various kinds, held together by mucus. The organisms are those usually found in the mouth; sometimes we find pathogenic organisms, most frequently the pneumococcus and the pus-producers. Parts of the papillæ are also found, depending largely upon the force used in scraping off the tongue. With the exception of the pathogenic organisms, then, nothing specific is found in this fur, and it would be futile to attempt to speak of any specific coating for any given disease, on the basis of what goes to make up this coating. But if we go one step farther, it will be seen how a general process may be followed by the same process upon the tongue. We abstract entirely from those conditions, like scarlatina, the geographical disease, or syphilis, in which a definite local process is always followed by a well-specified appearance, which can be looked upon as characteristic, although I must confess to having seen a strawberry tongue in several instances without scarlatina. Three things are requisite in order that the mucous membrane which covers the tongue shall be in its normal condition,—moisture, a proper nutrition for the epithelial coating, and sufficient motion. Anything which affects either of these three factors will cause some change in the covering of the tongue. If, in

diseased conditions, there be added those causes which produce a deposit of any foreign material, such as coloring matters, in the coating of the tongue, we have all the elements required for explaining the various kinds of fur. Flat epithelium, as one of the lowest types of tissue in the body, is very easily affected by any slight deviation from its normal nutrition. The epithelium found upon the tongue is more or less opaque, depending upon the distance it is removed from the cavity of the mouth: the lower layers of cells, the younger ones, are translucent; the older ones have what has been called a more granular structure. The greater the number of the latter the thicker the fur; the greater the number of the former the thinner. When anything occurs to hasten the change from young to old, so that there are a great many more old, opaque cells than normal, the tongue will be furred. When, on the other hand, anything occurs to prevent this change or to materially retard the formation of epithelium, the tongue will be without fur and will seem red. The effect of moisture is in two directions: first, upon the appearance of the cells, and, secondly, upon their removal. When there is too much moisture in the mouth, the cells are short-lived and easily become converted from young to old, so that there is a furred tongue. When there is too little moisture, the cells remain too long upon the dorsum of the tongue, and therefore the tongue will be furred. An example of the former condition is found in the furred tongue of salivation, of the latter in mouth-breathers. In long-continued fevers, in which the absence of moisture is the predominating cause, we have a peculiar condition of dry white or yellow fur, quite thick and adherent. When this is removed, in the course of the disease, there is left a glistening, dry tongue, without very much fur,—the latter condition due, however, to a lack of nutrition, so that the lower layers of epithelial cells are not supplied in adequate numbers.

When there is not sufficient movement of the tongue there results a fur, because fewer of the old cells are removed than would be under normal circumstances. In paralytics we constantly see a furred tongue; in any condition in which sensation is obtunded—high fevers, soporose or comatose states—the same will be observed. These three factors, combined with the rest, produce the dry, coated tongue of typhoid conditions, which finally result in cracks of the whole mucous membrane, giving rise to small hemorrhages, which give to the tongue a brown or reddish-brown color.

The supply of nutrition to the epithelial cells is of importance, in that the cells that grow old have to be replaced by young ones. When this cannot be done, no fur is produced, but the tongue has a red appearance; and if the coating be examined under the microscope, few adult cells are found. We find this condition, especially, in long-continued disturbances of general nutrition, in adults in cancer, in children in paedatrophia or long-continued chronic intestinal catarrh. A supply of too much nutritive material, overfilling of the lymph-spaces from too abundant blood-supply, acts very much like too much moisture. The cells are hastened in their course of life, too many older ones are produced, and there results furring. The place of deposit of this fur depends very much upon the size and shape of the tongue; where the tongue does not come in contact with any other part of the mouth it will be thick, at the edge it will be rubbed off, leaving a red outline. This is the character of cyanotic tongues, especially pertussis. In fevers the amount of nutritive material supplied plays a very important *rôle*; but we can only repeat what we have stated before, that there is no necessity for the production of the classical typhoid tongue, with crusts and fissures, as this can be readily prevented by supplying the factor of moisture.

Great stress has always been laid upon the foreign admixtures; especially to the coloring matters. For instance, a

peculiar coating of tongue has been accepted as characteristic of malarial troubles,—a yellow coating at the base of the tongue. A yellowish tongue has always been associated with liver troubles, and has been followed up by a dose of calomel. There are many pigments that will produce a yellow color besides bile-coloring matter, both from within and upon the tongue, and a diagnosis of biliousness, which means nothing, is on a level with the practitioner who is willing to prescribe by looking at the tongue only. If we take into consideration that in that form of trouble in which we know that bilirubin is in the circulation—jaundice—we frequently find the tongue clean (Henoch), often white, and rarely yellow, we certainly must be careful in drawing the conclusion that because the tongue is yellow the liver is at fault. It is just as probable that some chromogenic organisms or some extraneous substance is the cause of a yellow tongue. Every one has seen patients who are never without a slight yellow fur and yet seem to enjoy perfect health. The most ludicrous mistakes occur to those who overlook the fact that articles of food and medicinal agents give their color to the fur; rhubarb produces a beautiful liver tongue. Deposits of pigment in the mucous membrane of the tongue are of much greater diagnostic value. The black pigments of melanosis, the malarial cachexia, or Addison's disease, do much to draw the attention of the physician in the right direction.

Ulcers upon the tongue have been described in other chapters. There is one form of ulceration which, from time to time, is described as a new discovery and considered as a pathognomonic sign for whooping-cough. It is a symptom that has been noticed by a great many of the comparatively older writers; indeed, no complete description of whooping-cough could be written without its mention, but it is not to be looked upon as pathognomonic. In certain conditions, when a child has a long-continued cough, there appears first a cloudi-

ness of the frenulum linguæ, which is followed by a loss of substance more or less deep. This ulcer cannot be produced unless the child has its two central lower incisors, and occurs only in violent, persistent coughs in which the tongue is forced out of the mouth. When the cough is very severe and the child has its lateral as well as its central incisors, that part of the tongue which, in coughing, is forced and rubbed over these teeth may also become ulcerated. This ulcer of the frenulum has been seen by a great many authors in coughs that were not pertussis, and I can add my own testimony to the correctness of this observation. It will be found in bronchitis; sometimes, but rarely, in pneumonia; more commonly in the cough associated with enlargement of the bronchial glands. The appearance of the tongue, as a whole, the coating, and the cloudiness or ulceration of the frenulum linguæ are very valuable aids to the early diagnosis of whooping-cough.

The mouth plays a very important *rôle* in the differential diagnosis of the acute exanthemata. On account of the fact that the eruption makes its appearance in the mouth in from one to two days before developing upon the skin, valuable knowledge can be gained by careful attention to the mucous membrane. Especially is this the case in the early, differential diagnosis between measles and scarlatina. The changes in the tongue, in measles, have already been referred to. In the majority of cases of measles, at least forty-eight hours before any eruption is to be seen about the face, we can observe a decided reddening of the posterior pillars of the fauces, and with this a small reddish, or reddish-blue, papular eruption upon the soft palate, hemorrhagic in hemorrhagic measles. This lasts a few days, disappears, and not infrequently leaves pigmented spots. In scarlatina, the anterior pillars of the fauces and the tonsils are first reddened, and this is followed, in a very short time, by the appearance of the eruption, in the form of a bright red erythema, upon the

centre of the soft palate. From here it extends, sometimes developing over the posterior part of the hard palate, sometimes over the whole mouth; the greatest development of this eruption is arrived at before the rash develops upon the skin. Although the whole mouth may remain red, during the early course of the disease it is more diffuse, not so punctate and not so bright. Hand in hand with this goes the development of the so-called strawberry tongue. At first the tongue is covered with a milky fur; very soon, however, the papillæ, especially the fungiform papillæ, become enlarged and very prominent, the white fur begins to disappear, first about the edges and then towards the centre, and we finally have a tongue deprived of all fur, with the filiform papillæ apparently gone, but the fungiform very prominent, giving in all the characteristic tongue of scarlatina. This may be absent in very mild cases, and, again, may be present in other conditions besides scarlatina. Both of these occurrences are so rare, however, that they may almost be left out of consideration. In variola the erythema begins upon the posterior wall of the pharynx, and upon this, in a short time, there are developed papules, which, in their turn, are rapidly converted into pustules. In variola, the whole mouth participates in the process, and we see pustules upon the soft palate, the uvula, the tonsils, the hard palate, the cheeks, and the tongue. All this is not uncommonly accompanied by more or less salivation, swelling of the mucous membrane, and enlargement of the tongue. In varicella we never see any of the preparatory stages, but always the pustule or small ulcers which are left where these pustules have existed. My experience has been opposed to that of Löri, who says that pustules are rarely developed upon the mucous membrane. I have rarely seen a case of varicella in which there could not be found one or more pustules in the mouth or in the pharynx. These changes, briefly described, have been of the greatest assistance

to me in the early differential diagnosis of the acute exanthemata. Especially has this been the case in colored children. The diagnosis of scarlatina, in a full-blooded negro child, becomes almost impossible when the changes in the mouth are not taken into consideration.

The appearance and movements of the tongue are very much affected by lesions of the nervous system. In paralytics one-half or both halves of the tongue may be affected. When one-half is affected, as the result of a cerebral lesion, motion and even nutrition become changed, the paralyzed side becomes smaller, and the tongue, when in the mouth, deviates to the healthy side; when protruded, to the paralyzed side. In children, it is especially post-diphtheritic paralysis that affects the tongue. Disturbances in the nerves leading to the tongue may also produce paralysis, but this is very rare in children. Labio-glosso-laryngeal paralysis is a disease of later life, and, therefore, does not play a very important *rôle* in the semeiology of the mouth.

X.

PAROTITIS.

INFLAMMATIONS of the parotid gland are due to the localization of some morbific agent within the gland substance, which is usually of a general systemic nature. We exclude here those forms of parotitis due to trauma, and we see any number of general disease-producers affecting the parotid gland. A great many attempts have been made at classifying the various forms of parotitis; all of which, however, are more or less unsatisfactory on account of the purely pathological basis which underlies them. Any classification must, of necessity, be incomplete until the specific cause or causes of inflammation of the parotid gland shall have been discovered. For the present we are justified in making a clinical division only, with the reservation that future discoveries may make a great many subdivisions. Indeed, from clinical observation, it is possible to reason out more than two kinds of parotitis; but, as we have only probabilities and uncertainty to deal with, it seems wiser to defer these hair-splittings until the subject can be worked out from the proper stand-point. For our present purpose a division into primary and secondary parotitis will be sufficient. By primary parotitis is meant that form of inflammation of the parotid which develops without the intervention of any other cause than the one producing this inflammation; by the secondary form is meant that inflammation following or accompanying some other disease, in which it is rational to suppose that the poison producing this disease also causes the parotitis. Under the first heading is found, especially, epidemic parotitis, either in its epidemic or sporadic form, mumps; and, secondly, that rare form of parotitis due to an extension of an inflammation from the mouth to the parotid gland, by way of Steno's duct.

XI.

EPIDEMIC PAROTITIS (MUMPS).

THIS disease was thoroughly well understood by the ancients, Hippocrates, Celsus, Aetius, Galen, and others, and they described it just as it would be described to-day, even as far as the complications are concerned. There is, then, no historical development of the subject; indeed, it might be said that very little new has been added since the days of Hippocrates, and if anything new will be added it will be in the direction of the etiology of the disease, concerning which we are completely in the dark. If we are permitted to judge by analogy, we are forced to class mumps with the acute infectious diseases: it has a period of incubation, one of invasion; the disease runs its course in a self-limited way; it is contagious, and the same individual is subjected to only one attack. The cause of the disease has not been discovered; but, as far as we know, it is a poison that is not very virulent; it may limit itself to a city, to a village, or even to one institution (a children's hospital, an orphan asylum) in a place without spreading, notwithstanding the fact that no precautions are ever taken to prevent its spread. Again, it may spread from one of these places of infection, so that it becomes quite general. On the other hand, sporadic cases of mumps are not so uncommon, and with a very slight degree of precaution, such as would not at all influence the spread of scarlatina, measles, or pertussis, these cases can be made to remain sporadic. The disease is found in all latitudes, and, as far as we can ascertain, in all countries. Some regions remain untouched for years, then several successive epidemics will occur, nobody seeming to know whence they come, and then these regions may remain exempt again for years. Or, in large cities, sporadic cases may occur at all times, and suddenly an epidemic may develop. In large clinics mumps may be observed at almost

any time of the year. The statistics of Hirsch and Leichtenstern seem to prove that the disease is most common in fall and in winter ; this must be explained upon the same principle that influences the occurrence of measles, scarlatina, pertussis, diphtheria, etc. In fall and in winter children are more apt to be kept in the house than in spring and summer ; if they come in contact with other children it will be very directly, in rooms. In spring and summer they are out of doors, and the contact there is more or less indirect, at all events in the open air, and contagion is not so likely to be carried.

Much stress has been laid upon the weather as an etiological factor in the production of the disease ; cold, damp, rough weather predisposing to epidemics. Although this may be true, the direct relation between mumps and the weather has not, as yet, been discovered, and when the cause of the disease is isolated, it will probably be found that some other reason can be assigned to this apparent connection.

It would be idle to discuss the nature of the poison any further ; this has been done very extensively, and the conclusions arrived at have differed somewhat. All that we know is how the invasion of a human being by this poison affects that human being, and that has been known since the days of Hippocrates. The poison is probably taken up by the mouth, and reaches the gland through Steno's duct, to have more or less effect upon the general system. This, again, is purely hypothetical, although we have very many poisons that act upon the general system which act in the same way,—the poison of typhoid fever, of diphtheria, measles, scarlatina, etc. For typhus fever and diphtheria this condition has been proven ; in mumps we have a period of incubation ; then more or less general symptoms ; a period of invasion, when, after these, the local manifestation of the poison begins, to be followed by local development of the poison in remote parts. This certainly looks as if the poison first taken into the gland multi-

plies there; during its biological activity in the gland produces a something which affects the general system to produce the general symptoms. Again, this poison may be deposited in other places and develop there as it did originally.

Children are most commonly affected between the ages of three and five years (Barthez and Rilliet); the disease is very rare before this time, and almost unheard of in very old people. Here, again, we have the general law of acute infections complied with. The disease is of very rare occurrence in infants, although it does occur in them. The fact, however, that a physician has seen a great many cases of mumps in infants should always lead one to doubt his diagnosis; indeed, for a simple matter, there are very few diseases in which so many errors of diagnosis are made as in mumps. It is further claimed that males are especially predisposed to epidemic parotitis; statistical proofs are too meagre to prove or disprove this assertion. As occupation can have nothing to do with predisposition in this disease, it is difficult to understand why one sex should be more favored than the other. As long as the catching-cold theory held full sway in etiology the most curious statements were to be found in the books; indeed, very few have, as yet, fully emancipated themselves from the cold and moisture theory in specific diseases. All of these statements, however true they may be, must await the future for credence; at the present they have been discarded.

The duration of an epidemic varies very much. Sometimes it is months, at other times a year or more, as has been intimated before; mumps rarely dies out in large cities. The number of cases existing in a place at a given time cannot be estimated except by direct count, as mumps does not figure in mortality tables. It is, therefore, difficult to state positively that a large place is ever free from mumps. Sporadic cases are undoubtedly due to the same poison as the epidemic cases. Why these sporadic cases do not give rise to epidemics it is

impossible to say. That the predisposition is removed by one attack can be urged for a certain number of individuals; but a good many individuals never get the mumps, even when exposed, and frequently a family, into which the disease has been introduced, is spared the first time, to be attacked by a subsequent exposure.

The period of incubation varies very much, according to different observers, in different epidemics. As low as from three to four days to the other extreme of twenty-five days has been observed. Leichtenstern places the period of incubation as lasting from seven to fourteen days; Vogel-Biedert, nine to twenty-five days; Rilliet and Lombard, in an epidemic at Geneva, as from twenty to twenty-two days. It is difficult to fix the period of incubation for any of the infectious diseases, and it is more than probable that this period differs in different individuals. It would be decidedly exceptional, however, if any disease could vary so much in its effects upon the individual as, in one instance, to take three days only to be followed by an effect, while in the other it would take twenty-nine days. It would seem most likely that we are dealing with an error of observation, which would be more than excusable in a disease like mumps, in which it might become impossible to localize exactly the source of infection.

On account of the fact that post-mortem examinations in mumps are so exceedingly rare, our knowledge concerning the pathological anatomy of the disease is very limited. Three views have been advanced: first, that the process is essentially a catarrhal one due to an inflammation arising in the salivary ducts and extending to the lining of the acini; secondly, that the inflammation arises and is limited in the large lymphatic spaces around the acini of the gland; and thirdly, that the inflammation is parenchymatous as well as interstitial. The second view has given rise to the term periparotitis, which was in vogue for a long time, but has now been discarded.

The only absolute evidence (Bamberger) that exists is in favor of the last view, but there are several considerations that must be taken into account before it is accepted. The histology of the parotid gland has been worked out since Bamberger published his article in *Virchow's Handbuch d. Spec. Path. u. Therapie*, 1855, and it is more than likely that, microscopically, the pictures obtained by him would be explained differently. Furthermore, the investigations of Heidenhain have placed the parotid gland in a class altogether different from the submaxillary and sublingual glands, in that it is different in its structure, its physiological activity, and its nerve-supply. While the fact is accepted that all the salivary glands are sometimes affected by mumps, yet this is exceptional, and the differences in all respects between them may possibly be the reason why they are not always affected together. The inflammatory process, as a rule, terminates in resolution, but the old dogmatic statement, "suppuration, no mumps," is not founded upon correct observation. On the other hand, the suppurative cases of mumps are so exceedingly rare that they are to be looked upon almost like the recoveries from tubercular meningitis. A curious fact to be noted is that the secretion of saliva is very little interfered with. If we would take the trouble to examine the saliva from the affected gland, we might find some changes, although the observations of Gerhardt and Lombard seem to contradict this. It is hardly possible that such extensive alterations in the gland tissue, even if they be but interstitial, should not be followed by some functional alteration; the more so is this the case when we know how little, in an experimental way, suffices to change the secretion. It is therefore more than probable that the saliva that has been examined is mixed saliva, the combined result of secretion from the other glands, and, as one of them is a mixed gland,—*i.e.*, both serous and mucous,—even if both parotids were affected, a difference could not be readily

detected, or that part of the gland unaffected produce sufficient saliva to cause the digestive changes.

As we are dealing with an acute infectious disease, the symptoms vary as they do in all of this class, depending upon the nature of the epidemic and upon the individual attacked. We can put down as the normal course of the disease about the following: the stage of invasion lasts from twenty-four to seventy-two hours; the local symptoms from eight to twelve or thirteen days, during which time the complications set in which may cause an indefinite sickness; but, upon the whole, the length of a normal attack, uncomplicated, can be put down as running from ten to fourteen days. In very mild epidemics the prodromal stage causes so few symptoms that it is overlooked. In very severe epidemics we have all the symptoms of malaise, more or less fever, as high as 104° Fahrenheit in the evening, sometimes vomiting and diarrhoea, and, in irritable children, so-called brain-symptoms; twitching, restlessness during sleep, talking and crying out during sleep, vomiting, convulsions, with contracted, dilated, or unequally-contracted or dilated, pupils. With the beginning of the local symptoms all of these general disturbances usually disappear. The first local symptom complained of is usually pain in a space between the mastoid process and the lobe of the ear; very soon this painful spot increases in size until the whole region around the ear, frequently the ear itself, and the whole side of the head become affected. Movement of the masseters, as in chewing, increases and promotes painful attacks, and in very mild attacks this is the only pain that is complained of by the patient. As a rule, the swelling begins in the same place where the pain is first noticed, to become general after from twelve to thirty-six hours. Upon this swelling and upon its accurate observation depend the accuracy of our diagnosis. The fact must not be lost sight of that the parotid gland, as its name implies, lies *around* the ear. There is a lymphatic gland

that lies within or upon the parotid gland ; there are lymphatic glands that lie behind, and others that lie below the ear ; all of these may swell, and many a case of mumps is nothing more nor less than a swelling of one of these glands. There is but one gland that lies around the ear,—*i.e.*, in front, following the general outline of the ear, below and behind,—and when the swelling is localized in this general outline, we are dealing with one thing and one thing only,—parotitis. From a point between the lobule of the ear and where the mastoid process should be the swelling extends backward around and forward around, and in mild cases is limited to this general contour. In some cases the swelling extends upward towards the orbit ; in most cases it extends to the temporal region. Downward, it may go along the neck, being limited for anatomical reasons by the clavicle. All this swelling causes a peculiar appearance, but the effect upon the ear is especially characteristic. The swelling causes the ear, as a whole, to be shoved away from the side of the face, but on account of the fact that the lobule is the most movable part of the auricle, it is most apparent there. Indeed, the upper part of the auricle seems nearer to the face than under normal conditions, due to the swelling, while the lobule is turned up, pointing either forward or backward, and rotated, as a whole, slightly upon its horizontal axis.

Heretofore we have seen the superficial swelling only ; in some cases the process attacks deeper parts, producing dysphagia, and causing pharyngitis, laryngitis, and oedema of the glottis. The internal swelling, in double mumps, may become so great as to prevent swallowing entirely, and then we have the aspect of a very sick patient ; or the oedema of the glottis may become so great as to demand operative interference ; both conditions, however, are very rare, and much consolation may be derived from the consideration that the acme of the process is attained very quickly and is very short-lived. In some

epidemics the submaxillary and sublingual glands are always enlarged; in others we find special lymphatic glands swollen. I have observed one epidemic in which the enlargement of the parotid gland seemed to be a secondary consideration, in that the principal swelling took place in a large lymphatic gland lying below and slightly in front of the parotid,—a gland belonging, probably, to the deep cervical chain. In this epidemic, the peculiarities of which were observed by several other physicians, the swelling began in this gland, was followed in a short time by a decided, though comparatively slight, enlargement of the parotid, and then ran its course in the usual way.

An examination of the patient reveals other facts. The lymphatic glands may be enlarged,—the axillary, the inguinal, and the cervical; but not much reliance can be placed upon this symptom: first, because this enlargement is common to nearly all acute infectious diseases; secondly, because it is impossible, in any individual case, to state that the lymphatic glands are not of a normal size for that individual, or have not become enlarged from some other cause. It is claimed that, in a great many cases, the spleen also is enlarged; in another place ("Malaria," Keating's "Cyclopaedia," vol. i.) I have pointed out the difficulties that beset this diagnosis. I have sometimes thought that the spleen was enlarged, but certainly not often enough, nor constantly enough, to have made this symptom of any importance in the way of helping to make an early diagnosis.

The patient presents an almost comical appearance when the swelling has arrived at its maximum. He holds his head stiff, usually inclined towards the affected side; if both parotids are swollen, the head is held like a patient having cervical vertebral caries. The face is swollen; if unilateral, one side presents an altogether different appearance from the other; if bilateral, we frequently find the circumference of

the face much greater than that of the head. On account of the swelling, the play of the facial muscles is interfered with and the expression of the face becomes set; even laughing or crying may hurt so much that the patient becomes very quiet. The folds of the face, if there be any, are obliterated, and the natural depressions no longer exist; in this way the deformity may become great. In these cases the tongue and mouth become coated and foul; catarrhal or other forms of stomatitis may develop. The swelling itself is doughy, very painful, the skin covering the gland tense but anaemic; when it becomes red, it is usually presumptive evidence that mixed infection has taken place; in other words, that some other virus besides that of mumps is flourishing in the infected tissue. Besides the local pain, it will be found that the patient complains most of the difficulty in swallowing, but this mechanical disturbance may extend to the other organs; the ears sometimes become affected; the patient complains of tinnitus, shooting pains in the ears, slight deafness, and, rarely, middle-ear trouble may arise from a case of mumps.

Bilateral affection is common, possibly the rule; but this differs with the character of the epidemic. In some epidemics nearly all the cases are bilateral; in others, again, very few are found in which both glands become affected. As a rule, both glands are not attacked simultaneously; the one begins and is followed, in a few days, by swelling of the second. The swelling in the second gland does not attain the same degree of intensity as in the first, although this is subject to exceptions.

The attempt has been made to characterize the fever-curve of this disease; but variations are so very common that nothing typical can be recognized. The more intense the infection the higher the temperature; the maximum may be observed during the period of invasion, to decline gradually during from five to seven days, until a normal evening temperature is attained, and to show exacerbations with the development of any com-

plication. Affection of the second gland, for instance, always produces a rise in temperature. Close observation will establish the fact that all cases of mumps are attended with more or less fever; the rise may be very slight, but it will be found, especially in the evening. I have paid especial attention to this point, and have never, as yet, found a single case in which, at some time or other, there has not been a rise in temperature. From a theoretical stand-point, afebrile cases of mumps should exist, and further observation may, probably, establish their existence. I have never observed any case in which the maximum was much over 104° Fahrenheit, although some cases are on record in which this maximum has been exceeded. In some epidemics we find very low temperatures, in others we find few cases in which the temperature does not mount up; we no longer look upon the degree of fever as meaning direct danger to the patient, yet Debize (quoted by Leichtenstern, Gerhardt's *Handbuch f. Kinderkr.*, ii. p. 665) speaks of cases in which the temperature remained in the neighborhood of 104° Fahrenheit for several days, when a typhoid condition developed accompanied by "prostration, apathy, somnolence, delirium, dry fuliginous tongue and mouth." It has not been my lot to see such cases, and, having seen a very great number of cases of mumps, I am almost inclined to suspect that there was some other reason for this combination of symptoms than simple epidemic parotitis; the more so, as the author reports more than one case. The pulse does not present anything characteristic, usually following the course of the temperature.

The affection, without complications, and even with most of those that are not extremely exceptional, can be looked upon as very trivial in nature. The fact that so little is known concerning its etiology shows how very rare mortality is; as to its sequelæ, more will have to be said in the future. As a rule, an attack is terminated after the glands have undergone the changes described before. The whole process will last

from one to two weeks, and, in the great majority of instances, there is complete *restitutio ad integrum*. Cases will occur in which the duration of the disease may be somewhat longer, and some epidemics have been described in which there occurred veritable relapses.

The termination in suppuration is an extremely rare one. The fact that very many authors have never seen suppuration in mumps does not put us in the position of being able to reject the evidence that is given with great precision by other writers. Every one admits that this complication is an exceedingly rare one, and, from the reports that can be looked upon as authentic, it seems that the formation of abscesses in mumps occurs in particular epidemics. The author has never seen suppuration, and has always looked upon those who have seen a great number of cases as having been misled by changes in glands near the parotid. Indeed, in all those cases which he has seen in which suppurating parotitis was claimed, it has been possible to prove that the process was not in the parotid but in lymphatic glands, lying either upon or below the parotid gland. A case of this kind is reported by Barthez and Rilliet, and greater care in localization would undoubtedly confirm the truth of the above statement. From a review of the literature suppuration after parotitis epidemics must, however, be accepted as a fact, and possibly the next epidemic that appears will bring us cases of this description.

The most common complication of mumps in adults is orchitis or epididymitis. In children this complication is rarer, judging by the number of recorded cases, than suppuration. The author has seen one case, in a boy six years of age, that ran its course in the same way as in the adult. Other cases are reported by De Lens, Wolff, Demme, and Homén. Why this complication should arise so commonly in adolescents and adults and not in children it is difficult to say, but it seems almost impossible to exclude the functional activity

in the adult as being one of the predisposing causes. That the orchitis is due to a localization of the mumps virus can be taken for granted in comparing the local process of mumps with that of other infectious processes.

Other complications have been recorded : albuminuria, paralysis, such as may follow any infectious process, and troubles with the ear. The latter seem of special importance, as they may lead to complete deafness and therefore to deaf-mutism.

Upon the whole, the prognosis is universally a favorable one. Exceptionally, a patient may die from œdema of the glottis, burrowing of pus, or one of the general complications, but all this is so rare that, practically, it may be neglected.

The treatment is what might be called a typical expectant one. We do not know anything about the poison, we have no remedies that affect the process, and the complications are rarely such as require interference. It is the custom to cover up the affected gland with cotton and to rub some oily substance into the skin. Both are unnecessary, but inunctions sometimes give relief to the patient, and the application of anything externally gives comfort to the surroundings. If complications arise they must be treated as such.

INDEX.

A.

Abscess, retro-pharyngeal, 20.
Acarus scabiei, 154.
Acid, boric, 31, 66.
 carbolic, 104.
 hydrochloric, 96, 97.
 nitric, 96.
 osmic, 51.
 pyroligneous, 97.
 tannic, 122.
Acne vulgaris, 23.
Ætius, 126, 180.
Albrecht, 115.
Alkalies, 52.
Almonds, oil of sweet, 157.
Alveolus, 131.
Amulets, 125, 126, 127.
Amygdalitis, 60.
Anderson, 166.
Angina crouposa, 100.
Antiseptics, intestinal, 156.
Aphthæ, 9, 34, 82, 83.
 Bednar's, 43.
Aphthous sore throat, 19.
Aristotle, 126.
Armstrong, 129.
Arsenic, 17.
Ashley, 159.
Astringent, 122.
Athrepsia, 47.
Avicenna, 46, 127.

B.

Babes, 89.
Baginsky, 42, 49, 56, 67, 101, 104.
Bamberger, 184.
Bärensprung, 37.
Barlow, 83.
Barthez, 68, 71, 73, 89, 96, 129, 136,
 158, 166, 182.
Bath, lukewarm, 155.
Bednar, 43, 45, 129.
Bednar's aphthæ, 43.
Behrend, 165, 166.
Berg, 47.
Bergeron, 73, 75.
Berker, 109.
Biedert, Vogel-, 183.
Billard, 33.
Billroth, 35.
Birch-Hirschfeld, 53.
Blanchet, 47.
Bockhardt, 42.
Boerhaave, 46.
Bohn, 28, 33, 34, 35, 36, 37, 41, 43,
 47, 61, 68, 71, 72, 73, 76, 82, 89,
 92, 97.
Bôkai, 20.
Bonney, 166.
Borax, 66.
Borax-and-honey mixture, 66.
Bouchut, 115, 116, 129.
Bretonneau, 9, 47, 68.

Breveld, 48.
 Bromides, 155.
 Bronchitis in connection with teeth-ing, 153.
 its effect on the color of the tongue, 171.
 Buck, 21.
 Butlin, 109.

C.

Cairns, 162, 167.
 Calomel, 67, 155.
 Cameron, 36.
 Cancer, its effect upon the tongue, 174.
 Cancrum oris, 83, 87, 88.
 Caries, 77.
 Cartwright, Hamilton, 159.
 Castle, A. C., 166.
 Catarrh, chronic intestinal, its effect upon the tongue, 174.
 Catechu, tinctura, 122.
 Cauterization, 85.
 Cautery, Pacquelin, 96.
 Celsius, 180.
 Churchill, 166.
 Clarke, W. Fairlie, 20.
 Cnyrim, 38.
 Cocaine, 42, 84.
 Condylomata lata, 107.
 Copper, 69.
 sulphate, 96.
 Cornil, 89.
 Corrosive sublimate, 49, 67, 104, 121.
 Croup, 99.
 Cyanosis, general, the color of the tongue as a symptom of, 71.
 Cysts, 26, 32.
 retention, 43.

D.

Day, 159.
 Debize, 189.
 Dentition, 124.

Dentitio difficilis, 125.
 Des Forges, 166.
 Diarrhoea, long-continued, its effect on dentition, 142.
 Diphtheria, 92, 99.
 of the mouth, primary, 100, 101.
 Disease, hoof-and-mouth, 38.
 Dorning, 136, 160.
 Dyspepsia, 16.
 Dysphagia as produced by parotitis, 186.

E.

Eczema, 153.
 ad natem, 62.
 as caused by stomatitis catarrhalis, 28.
 Emplastrum hydrargyri, 122.
 Enamel germ, 130.
 Epstein, 43, 55.
 Erosions, syphilitic, 106, 107.
 Erythema, 23.
 of the mouth, 23, 24.
 Eustachius, 127.
 Evanson, 96, 97.
 Excoriations, 121.

F.

Fevers, long-continued, their effect on dentition, 142.
 Finlayson, James, 156, 166.
 Fischer, 13.
 Fischl, 44, 45.
 Fissures, syphilitic, 106, 107.
 Fleischmann, L., 125, 126, 129, 131, 136, 140, 141, 156.
 Foerster, 97.
 Follicles, muciparous, 25.
 Food, defective, its effect on dentition, 136.
 Fossanagrives, 48.
 Fournier, 118.
 Freeman, 36.

<p>Fröhwald, 90. Fur on the tongue, the place of deposit, 174.</p> <p style="text-align: center;">G.</p> <p>Galen, 9, 46, 126, 180. Galvano-caustic wire, 96. Gangrene, 92, 93. Garland, J. W., 166. Gerhardt, 36, 73, 88, 138, 184. Germ, dentine, 130, 131. Gierke, 91, 92, 95, 97. Girtanner, 129. Glands, axillary, 187. cervical, 187. inguinal, in parotitis, 187. lymphatic, 27, 79, 81. as affected by parotitis, 187. sublingual, as affected by parotitis, 187. submaxillary, as affected by parotitis, 187. tuberculosis of, 21. Glossitis, the tongue in, 170. Glottis, œdema of the, as caused by parotitis, 186. Glycerin, 121. Gonococcus, 153. Grawitz, 48, 56. Grünfeld, 115. Gubler, 109. Gums, lancing of the, 127. Gustin, 100.</p> <p style="text-align: center;">H.</p> <p>Hæmatoidin, 23. Hall, Marshall, 156, 158, 159, 160, 161. Hamilton, 166. Hare, brains of, 157. Haussman, 54. Hebra, 125.</p>	<p>Heidenhain, 184. Heliotrope, decoction of, 127. Hemorrhage, its effect upon the tongue, 171. Henoch, 71, 73, 175. Heredity, its effect on dentition, 136, 142. Herpes, 37, 153. Hippocrates, 9, 33, 46, 126, 180, 181. Hirsch, 73, 89, 181. Hirschfeld, Birch-, 53. Hodgkin's disease, the tongue as seen in, 171. Honey, 157. Hunt, 14. Hunter, John, 128, 156, 158. Hutchinson, Jonathan, 114, 115, 116, 117, 118, 119. Hydrocephalus, 140. Hyperæmia, 23. venous, 24. Hyposulphites, 66.</p>
	I.
	<p>Icterus neonatorum, 23. Idiocy, premature teething in connection with, 139, 140. Intertrigo, 62. Iodine, 69, 71. Iron, white-hot, 96. persists of, 104.</p>
	J.
	<p>Jaborandi, 11. Jacobi, 14, 101, 103, 129, 136, 139, 160. Jaederholm, 14. Jörg, 68.</p>
	K.
	<p>Kaposi, 37. Keratitis, 150.</p>

<p>Koch, 89. Korownin, 10.</p> <p style="text-align: center;">L.</p> <p>Lancet, 160. Landerer, 15. Lanolin, 122. Laryngitis, as caused by parotitis, 186. Lassar, 75. Lead, 69, 71. Leeches, 129. Leichtenstern, 181, 183, 189. Leptothrix, 122, 123. Lichen, 153. Lichenoid condition, 109. Lingard, 87, 90. Linossier, 49. Lip, hare-, 22. Listerine, 85, 120. Loeffler, 102. Lombard, 183, 184. Löri, 177. Lutschbeutel, 22.</p> <p style="text-align: center;">M.</p> <p>Magitot, 141. Magnesia, 47. Marchand, 14, 15. Marsh-mallow, 157. Maunsell, 96, 97. Measles, the tongue in, 23, 24, 171. Mercury, 69. bichloride (<i>see</i> Corrosive sublimate). Mering, 14, 15. Methæmoglobin, 14, 15. Micrococci, 91. Milia, 43. Miller, 126. Millet, 47. Minnich, 102.</p>	<p>Mischterlich, 11. Moisture, its effect upon the tongue, 173. M. Money, 159. Monilia candida, 49. Monti, 171. Mouth, the, as an aid to differential diagnosis in the acute exanthemata, 176, 177, 178. follicular sore, 19. gangrene of, 87. how to prevent affections of, during fever, 30. hyperæmia of, 23, 24. in varicella, 177. in variola, 177. of an infant, 10. how to cleanse, 30. Muguet, 46. Mumps, 180, 189. suppurative, 184. Mundfäule, 68. Mundschwämchen, 46. Mycelium, 58. Mycoderma vini, 48, 49, 58.</p> <p style="text-align: center;">N.</p> <p>Nägeli's fluid, No. 1, 51. Nationality, its effect on dentition, 136. Necrosis, 76, 80, 83, 85. tissue, 89. Nicati, 115. Nicol, 166. Noma, 83, 87, 88, 89.</p> <p style="text-align: center;">O.</p> <p>Odontopathie atrophique, 114. Odor, fetid, 78. Œsophagus, thrush of, 61. Oidium albicans, 21, 48. Opiates, 156.</p>
---	--

Orbicularis palpebrarum, 150.
Oribasius, 126.
Oxyhæmoglobin, 15.

P.

Pacquelin's thermo-cautery, 98.
Palate, cleft, 22.
Papayotin, 104.
Papillæ, filiform, 26.
fungiform, 26.
Papules, syphilitic, 106, 107.
Paré, Ambroise, 127, 156, 157.
Parotitis, epidemic, 179, 180.
primary, 179.
secondary, 179.
Parrot, 47, 53, 56, 109, 114, 118.
Paul of Ægina, 127.
Pemphigus, 153.
Pepsin, 17.
Periparotitis, 183.
Pharyngitis as caused by parotitis, 186.
Phlyctenular conjunctivitis, 150.
Phosphorus, 69, 71.
Pierce, 132.
Plant, 49, 56.
Plaques muqueuses, 107.
syphilitic, 106.
Plenk, Jacob, 128.
Pneumonia, catarrhal, 92.
Politzer, 129.
Potassium chlorate, 14, 15, 16, 22,
31, 42, 66, 84, 122.
poisoning by, 15, 16.
permanganate, 42, 66, 85, 97,
104, 120.
Poultices, 157.
Process, necrobiotic, 80.
Prophylaxis, 29, 65, 83, 120, 121.
Pruritus, 147.
Psoriasis, 109.
Ptyalin, 10, 11.

Puceron, 157.
Pyorrhœa, dental, 72.

Q.

Quince, 157.

R.

Rachitis, 118 (*see Rickets*).
Rajewsky, 14, 56, 102.
Ranke, 87, 88, 89, 90, 91.
Ratanhiaæ, tinctura, 122.
Reess, 49.
Rehn, 83, 137.
Resorcin, 67.
Rhagades, 106, 107, 121.
Rhazes, 127.
Rhubarb, 47, 155.
Richardson, B. W., 166.
Rickets, 114.
its effect on dentition, 141, 142.
Ringworm, 109.
Robin, 48.
Rosen, 46.
Rosenstein, Rosen von, 48, 128.
Roux, G., 49.

S.

Sac, dental, 130, 131.
Saccharomyces albicans, 49, 51, 53,
55, 56, 58, 59, 61, 62, 64, 65, 67,
123.
Saliva, 78, 79, 84, 103.
from gland affected by mumps,
185.
in the newly-born, 10, 11, 12.
Salivation, 81, 84, 102, 120.
as affected by dentition, 145, 146.
in stomatitis catarrhalis, 27.
in stomatitis ulcerosa, 78, 81, 84.
Salol, 31, 66, 85.
Sanné, 100, 101, 103.
Sattler, 87.

Scarification of gums, 126, 129,
 156-162, 167.
 its effect on convulsions, 161.
 its effect on diarrhœa, 164.
 Schaefer, 131.
 Schizomycetes, pathogenic, 21.
 Schnitzer, 68.
 Schrakamp, 41.
 Scorbutus, 72.
 Sée, Prof. G., 101.
 Seitz, 101.
 Semple, 159.
 Sequestra, 77.
 Seux, 60.
 Sialagogues, 11.
 Silver nitrate, 31, 67, 85, 97, 104,
 121.
 Simon, 48.
 Smith, J. Lewis, 97, 136.
 Sodium, bicarbonate, 31, 66, 67.
 salicylate, 31, 120.
 Soor, 46.
 Sordes, 30.
 Starr, 136, 160.
 Steiner, 129.
 Stomacace, 68.
 Stomatitis, 17.
 aphthosa, 33.
 catarrhalis, 19, 23, 39, 56, 63,
 99, 102.
 diphtheritica, 99, 100.
 erysipelatosa, 17.
 erythematous, 22, 23, 24.
 follicular, 20, 23
 gangrenosa, 83, 87.
 leptothrichia, 122, 123.
 mercurialis, 69, 70, 71, 122, 188.
 mycosa, 45, 46.
 with parotitis, 188.
 scarlatinosa, 17.
 simple, 19.
 syphilitica, 105.
 Stomatitis ulcero-membranous, 68.
 ulcerosa, 15, 17, 40, 41, 68, 122.
 Stumpf, 49.
 Synostosis, premature, 140.
 Syphilide, desquamative, of the
 tongue, 109.
 Syphilis, 108, 109.
 hereditaria tarda, 117.
 infantile, 119.

T.

Tannin, 122.
 Taupin, 68, 71, 73.
 Taynton, 166.
 Teeth, axe-shaped, 114, 115.
 cup-shaped, 114, 115.
 cuspidated, 114, 115.
 Hutchinson's, 117.
 notched, 114, 115.
 premature, 139.
 primitive, 139.
 the, as producers of stoma-
 titis catarrhalis, 19.
 screw-driver, 118.
 sulciformed, 114, 115.
 syphilitic, 114-117.
 Teething impetigo, 153.
 its effect upon the bowels, 151,
 152.
 Temperature in mumps, 189.
 in stomatitis catarrhalis, 27.
 Thrombi, 94.
 Thrush, 33, 45, 46.
 Thymol, 85.
 Tinctura ratanhiae, 122.
 Tongue, the, appearance of, 178.
 as affected by cancer, 174.
 by hemorrhage, 171.
 by Hodgkin's disease,
 171.
 by intestinal catarrh,
 174.

Tongue, the, as affected by moisture, 173, 174.
 by movement, 172, 174, 178.
 by paedatrophia, 174.
 coating of, 26, 169, 172, 173, 174.
 in stomatitis catarrhalis, 26.
 color of, 169, 170-175.
 in bronchitis, 171.
 in disease, 175.
 in general cyanosis, 175.
 in measles, 24, 170.
 in pertussis, 24, 170.
 influenced by extraneous substances, 175.
 cyanotic, 174.
 deposits of pigment in, 175.
 geographical, 109.
 ichthyosis of, 109.
 in coma, 174.
 in high fevers, 174.
 in glossitis, 170.
 in infancy, 169.
 in long-continued fevers, 173.
 in paralyties, 174.
 when sensation is obtunded, 174.
 in stomatitis catarrhalis, 170.
 ulcerosa, 170.
 in the newly-born, 10.
 its shape, 169, 170.
 its size, 169, 170, 171.
 lichen of, 109.
 normal mucous membrane of, 172.
 strawberry, 172, 177.
 typical typhoid, 174.
 ulcers of, in bronchitis, 176.

Tongue, the, ulcers of, in pertussis, 175, 176.
 in pneumonia, 176.
 Troussseau, 54, 68.
 Trypsin, 104.
 Tuberculosis, 153.
 Tylosis, 109.

U.

Ulcers, catarrhal, 42.
 chronic, 42.
 syphilitic, 107, 108.

V.

Valleix, 47, 60.
 Van Wimperse, 47.
 Venesection, 124.
 Vesalius, 127.
 Virchow, 76.
 Vogel, 129, 136.
 Vogel-Biedert, 183.

W.

Wagner, E., 53, 59.
 Wandering rash, 109.
 Wendt, 68.
 West, 14, 68, 92, 96, 129, 159.
 Whitworth, 166.
 Wichmann, 129.
 Wolf, 68.
 Wright, 159.

Y.

Yale, 166.

Z.

Zenker, 53.
 Zinc sulphate, 31, 96.
 Zone, infiltrated, 91.
 Zweifel, 10.

Thomas's Medical Dictionary.

A COMPLETE PRONOUNCING MEDICAL DICTIONARY. Embracing the Terminology of Medicine and the kindred Sciences, with their Signification, Etymology, and Pronunciation. With an Appendix, comprising an Explanation of the Latin Terms and Phrases Occurring in Medicine, Anatomy, Pharmacy, etc.; together with the Necessary Directions for writing Latin Prescriptions, etc., etc. By JOSEPH THOMAS, M.D., LL.D., Author of the System of Pronunciation in Lippincott's "Pronouncing Gazetteer of the World" and "Pronouncing Dictionary of Biography and Mythology." On the basis of Thomas's "Comprehensive Pronouncing Medical Dictionary."

Imperial 8vo. 844 pages. Extra cloth, \$5.00; sheep, \$6.00.

"I have examined it with especial reference to the *materia medica* and therapeutics, and find it greatly in advance of anything of the kind we have had for a number of years. In fact, a need existed for just such a book, and I will certainly recommend it to my class as the best of its kind. Its unquestioned merits will soon supplant all others."—PROF. J. A. MCCORKLE, *Long Island College Hospital, Brooklyn, N. Y.*

"It is just the book for a medical or any other student, and it should be in the office of every physician. This dictionary supplies a place that has never been filled. I have looked it through and find all the new words that I have sought."—PROF. A. F. PATTON, *College of Physicians and Surgeons, Boston, Mass.*

"No better testimonial to the value of the work can be given than the following from Dr. J. Gibbon Hunt, the distinguished microscopist: 'To me the work is invaluable. I am astonished at its fulness of all recent words which the modern advance of medical science has introduced. Of course I cannot conceive the learning and great labor which could edit such a complete, thorough, and admirable volume.' We can add nothing further save that the book should be in the hands of every physician and every student in the land."—*St. Joseph Medical Herald.*

* * * For sale by all Booksellers, or will be sent by the Publishers, free of expense, on receipt of the price.

J. B. LIPPINCOTT COMPANY.

715-717 MARKET STREET,

• • • • • PHILADELPHIA, PA. • • • • •

United States Dispensatory.

New (16th) edition. Illustrated. Carefully revised and re-written by H. C. WOOD, M.D., LL.D., JOSEPH P. REMINGTON, Ph.M., F.C.S., SAMUEL P. SADTLER, Ph.D., F.C.S. Containing more than 800 pages of new material. Green title label distinguishes the binding of the sixteenth edition.

Cloth extra, \$7.00; best leather, raised bands, \$8.00; half Russia, raised bands, \$9.00. For facility of reference, Denison's Patent Index will be inserted for \$1.00 additional to these prices.

The sixteenth edition contains over 800 pages of new material, including the latest information about the mydriatic alkaloids, the new antipyretics, antiseptics, etc. The National Formulary has also been incorporated, and the gain to both authorities of having their valuable information connected, so that whilst working from a formula the operator may have a reference to the article on the subject in the Dispensatory, is one that every practical pharmacist will thoroughly appreciate. The work is recognized by the government of the United States as the standard work of reference, and is endorsed and universally used by colleges of medicine and pharmacy and State examining boards.

"The work is well worthy of appreciation on the part of all interested in the progress of medicine and pharmacy, and we bespeak for it not merely a place upon the bookshelf of every pharmacist, but a careful perusal, as embracing much that is important in the way of current information, and as containing valuable matter as a work of ready and convenient reference."—*Druggists' Journal*.

"The book is bound to have an enormous sale, as it is a positive necessity to all who wish a complete compendium of drugs and medicines."—*Minneapolis Medical Journal*.

"This is undoubtedly the most important edition of this voluminous and indispensable work yet issued: not because it is the latest, but because it has gathered within its capacious limits everything that is new in *materia medica* or *therapeutics, chemistry, and pharmaceutical research*."—*Philadelphia Clinical Record*.

"We commend this work as a most valuable addition not only to pharmaceutical literature, but to the medical profession as almost invaluable. Its literature, its chemistry, and its pharmacy are fully up to any similar work here or abroad of its kind, and the high standard of excellence in the past is only enhanced by the thoroughly reliable and trustworthy work of the present edition."—*Pharmaceutical Record*.

** For sale by all Booksellers, or will be sent by the Publishers, free of expense, on receipt of the price.

J. B. LIPPINCOTT COMPANY,

715-717 MARKET STREET,

- - - - - PHILADELPHIA, PA. - - - - -

THE EIGHTH EDITION OF
WOOD'S THERAPEUTICS:

Its Principles and Practice. By H. C. Wood, M.D., LL.D., Professor of Materia Medica and Therapeutics, and Clinical Professor of Diseases of the Nervous System, in the University of Pennsylvania. A Work on Medical Agencies, Drugs, and Poisons, with especial reference to the relations between Physiology and Clinical Medicine. Price in cloth binding, \$6.00; sheep binding, \$6.50.

REARRANGED, REWRITTEN, AND ENLARGED.

Scarcely three years have elapsed since the appearance of the seventh edition, yet the preparation of the present volume has necessitated a careful study by its author of more than seven hundred memoirs. In the present edition no revolutionary changes have been made comparable to those of the seventh revision, but great care has been exercised to see that every portion of the work has been thoroughly revised, and a number of the articles have been completely rewritten, while some new drugs have been noticed. Among those portions of the book which are practically new may be mentioned, as important, the whole subject of Anæsthetics, the articles upon Cocaine, Strophantus, Caffeine, Antipyrin, Antifebrin, Phenacetin, Hydrastine, Paraldehyd, Lead-Poisoning, etc. Among the absolutely new articles may be mentioned Sulphonal, Chloralamid, Aristol, and others.

"This book should be in the hands of all who wish a safe and reliable treatise on the subject of therapeutics."—*Richmond (Va.) Southern Clinic.*

"Although always a favorite for the conciseness of the text and the reliability of therapeutic teaching, in its new dress it has excelled itself, and is likely to hold its own against all rivals."—*Wilmington (N. C.) Med. Journal.*

"As a work of reference it will form a most valuable addition to the library of every member of the medical profession."—*Edinburgh Medical Journal.*

"Taken all in all, we have little hesitation in pronouncing this the most reliable work on therapeutics in the English language."—*Philadelphia Medical Times.*

For sale by all Booksellers. Sent by the Publishers, post-paid, on receipt of the price. J. B. Lippincott Company, 715 and 717 Market Street, Philadelphia.

AN ELEMENTARY TREATISE ON HUMAN ANATOMY.

By Joseph Leidy, M.D., Professor of Anatomy in the University of Pennsylvania, etc., etc. New (second) edition, rewritten and enlarged. Containing 495 illustrations. 8vo. Extra cloth, \$4.00; sheep, \$5.00.

In the preparation of this great work, Dr. Leidy has given special attention to those parts of the human body, a minute knowledge of which is essential to the successful practitioner of surgery and medicine. The names in most text-books are given in Latin; the author, however, has as far as possible used an English equivalent for such names, the Latin being given in footnotes. The illustrations are numerous and largely original, and prepared in the best style of the engraver's art. As most of the recent text-books of anatomy are very cumbersome, the condensation of this volume is a feature of great merit. The present edition (entirely rewritten) presents the ripe fruits of Dr. Leidy's experience of many years of successful labor as a teacher and as an original observer and discoverer in anatomical science, and the work will be everywhere recognized as the leading authority on the subjects of which it treats.

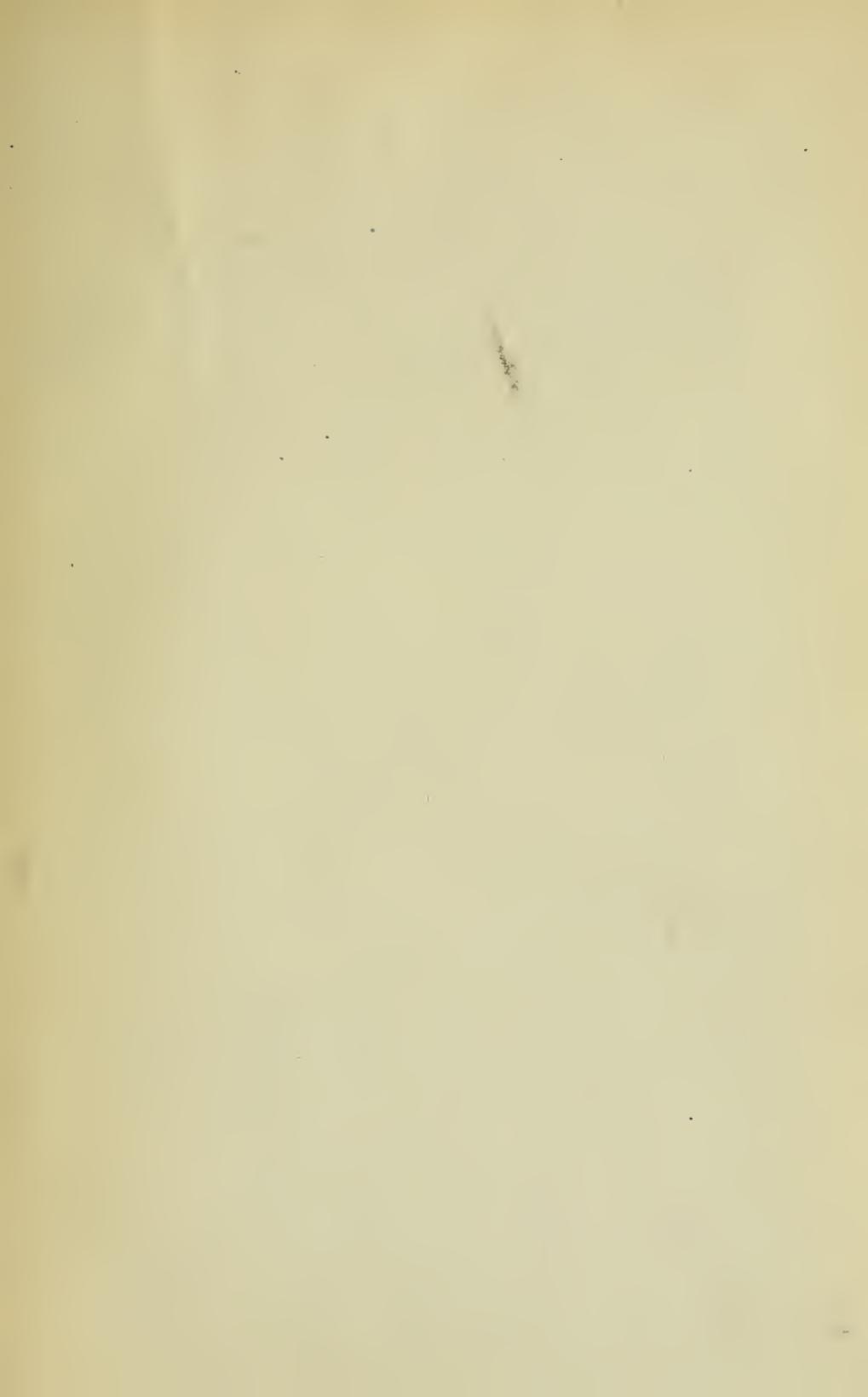
"After a thorough inspection I am pleased to pronounce 'Leidy's Anatomy' a most excellent work. It covers the entire field in a masterly manner, and deals with subjects entirely overlooked by other authors. It will afford me much pleasure to introduce it not only in my school, but to recommend it to the profession in general."—S. F. CARPENTER, *Northwest Medical College, St. Joseph, Mo.*

"The student can master and retain a practical knowledge of anatomy in a shorter time and with less hard work from this text-book than from any other work extant, and it has been our privilege to teach anatomy for several years."—*Ann Arbor (Mich.) Medical Advance.*

"We know of no book that could take its place, as it is written by a most distinguished anatomist. It has traits that no other work on the subject can boast of."—*St. Louis Medical Brief.*

* * * For sale by all Booksellers, or will be sent by the Publishers, free of expense, on receipt of the price.

J. B. LIPPINCOTT COMPANY,
715-717 MARKET STREET,
PHILADELPHIA, PA. - - - - -



Date Due

11-7-32

JAN 11 1939



RJ460

F77

Forchheimer

Diseases of the mouth in children

